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THE USE AND ABUSE OF THE ELECTROCARDIOGRAM IN MEDICAL PRACTICE*

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SINCE it is impossible to discuss briefly and adequately either the uses or the abuses of the electrocardiogram, my comments must be restricted to a few of the major aspects. Because the value of electrocardiography is more widely recognized than its limitations, I shall devote more attention to the latter. I shall try to consider matters that lie within the range of my personal knowledge and experience, or those that are accepted as established facts by acknowledged authorities in this field; any apparent dogmatism in my statements is due solely to limitations of time.

* * *

I shall consider very briefly a few of the more important ways in which electrocardiography can be of value in diagnosis and treatment; this discussion is not concerned with its advantages in laboratory research. I think one might mention first that this procedure is acknowledged to be the one certain method for the absolute identification of irregularities of the cardiac rhythm. It is true that electrocardiograms are often unnecessary for this purpose, for after several years of experience it is possible to identify most arrhythmias by means of auscultation alone. But there are circumstances in which clinical recognition is difficult or impossible, even for those with long experience, and at such times the electrocardiogram usually reveals the diagnosis immediately and clearly. If the arrhythmia is a complicated one, such as auricular fibrillation with a rapid ventricular rate complicated by frequent ectopic beats and short paroxysms of tachycardia, its certain identification is usually impossible without the aid of the electrocardiogram. Examples of this could be multiplied indefinitely, but the fact is too well established to require further discussion. This use of the electrocardiogram is possibly its most significant one in clinical medicine.

Furthermore, the electrocardiogram occasionally gives valuable indications that the heart has been involved in the course of systemic diseases. It is particularly useful in rheumatic fever, diphtheria and trichinosis; in any of these diseases, it may provide the first — and indeed the only — evidence that the infecting organism or the toxin has affected the heart. It is probably justifiable to say that every patient who has one of these conditions should have electrocardiograms taken at frequent intervals, at least during the acute phase of the disease.

Again, these graphic records are usually of great value in the recognition of acute myocardial infarction, which is due in most cases to coronary arterial thrombosis. They often make it possible to identify cases of old, and previously unrecognized, myocardial infarction, but these are usually less important because the necessity for immediate and careful treatment is less urgent. In most cases, the curves not only reveal that there is a fresh infarct in the wall of the heart, but also point fairly clearly to the exact area involved, although I question whether this is clinically so essential as it was considered several years ago. Blumgart and his colleagues[1] have shown that coronary thrombosis with or without myocardial infarction has already occurred in many patients whose histories indicate nothing more than mild or moderate anginal pain on effort. It is now known that the electrocardiograms of many patients who are just beginning to experience such pain display the characteristic and progressive changes of recent myocardial infarction, and thus provide clear indications for the proper treatment at the proper time.

Occasionally, the electrocardiogram reveals clear evidence of digitalis intoxication that was unsus-

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pected by careful and competent physicians. Although in some of these cases a critical review of the history indicates that overdosage should have been suspected, in others the dose has not been large and the usual symptoms of toxicity have been absent. The great significance of this diagnosis perhaps needs no emphasis, but it is well exemplified by a patient whom I saw recently: a sixty-five-year-old woman whose chief symptoms were physical weakness and profound depression, with occasional hallucinations and periods of disorientation. She had been in bed for several weeks, and the mental confusion was increasing; the possibility of admitting her to an institution for the treatment of mental disease was being seriously considered. She had taken digitalis for some weeks in doses of 0.1 gm. (1½ gr.) daily. Physical examination was entirely negative except for a third sound at the apex of the heart. The correct diagnosis — digitalis intoxication — was not suspected until the electrocardiogram was obtained; this showed incomplete auriculoventricular heart block, with a marked delay in auriculoventricular conduction, and depression of the RST segments. The third heart sound was due to the fact that the audible auricular contraction fell relatively early in diastole, and the mental symptoms were caused entirely by digitalis, since they disappeared completely after the drug was discontinued and reappeared in the same form when digitalis was resumed. This effect of digitalis is not especially uncommon in older people and is occasionally produced by relatively small doses, which may not affect the electrocardiogram so clearly as in this case. In another case, an excellent cardiologist made an unhesitating diagnosis of auricular fibrillation on the basis of auscultation of the heart when the condition was actually paroxysmal ventricular tachycardia, with alternation of upward and downward ventricular deflections due to severe digitalis poisoning. Fortunately, an electrocardiogram was obtained before the patient had received the large dose of digitalis that was prescribed. This mistake, which might easily have been made by anyone, emphasizes the necessity of securing these curves before proceeding with treatment, whenever it is feasible to do so.

Of less significance than any of the foregoing, but nevertheless not to be ignored completely, is the help that may be derived from the electrocardiogram in the recognition of structural lesions. Among the lesions that are sometimes reflected in changes in the curves are acute pericarditis and acute pulmonary infarction. I do not wish to place great emphasis on the role of the electrocardiogram in either of these conditions, because the changes are by no means constant and the diagnosis is often so clear from the clinical evidence alone that an electrocardiogram is unnecessary. Furthermore, there is not complete agreement as yet on the electrocardiographic pattern that should be accepted as indicative of pulmonary infarction, since this lesion may change the curves in a variety of ways. In chronic constrictive pericarditis, the electrocardiogram may provide evidence regarded by some as confirmatory, but it is not specific and the diagnosis should rest largely on other signs. Considerable emphasis has been placed on the presence of axis deviation of the electrocardiogram to the right or the left in the diagnosis of structural lesions that impose added work on the right or left ventricle. The classic association is that of right-axis deviation with mitral stenosis and of left-axis deviation with aortic-valve lesions or arterial hypertension. But these matters seem to me of relatively little consequence, since axis deviation may depend on many factors besides ventricular enlargement, and the diagnosis of valvular lesions should be made by auscultation without great difficulty and with considerable confidence.

There is time to mention only one other circumstance in which the electrocardiogram may prove to be of great value. Those who specialize in the field of cardiovascular disease often have occasion to see people who are disturbed and apprehensive because of their belief that they have heart disease, when as a matter of fact they do not. Sometimes, this fear is based on unimportant symptoms, such as slight superficial stabs of discomfort over the precordium or the consciousness of occasional premature beats; sometimes — and far too often — on injudicious comments by physicians or nurses. It is unnecessary to mention the more serious conditions known as neurocirculatory asthenia and cardiac neurosis. In the treatment of such patients, it is imperative to convince them that everything possible has been done to ascertain the condition of the heart. An electrocardiogram is an essential part of the examination of every such person, and it may be a comfort both to him and to the physician to know that it is a normal one. I do not for one moment forget the well-known fact that a normal electrocardiogram does not exclude heart disease and may actually be obtained from patients who have heart failure; also I do not forget that a normal electrocardiogram, when combined with a negative cardiac history and negative physical and fluoroscopic or x-ray examinations, is vital additional evidence that strengthens the physician's belief that the heart is free from serious disease. This may be of psychologic value only, but

it is psychologic treatment that such patients need. I have known many patients — among them, a considerable number of physicians — whose burden of fear and anxiety has been greatly lightened by the knowledge that the electrocardiogram was perfectly normal.

This very hasty and incomplete survey of the contributions that the electrocardiogram may make to clinical medicine might be summarized by the statement that it identifies with certainty all known irregularities of cardiac rhythm, it may provide the first sign that the heart has been invaded by disease, it often shows in unmistakable terms that myocardial infarction has occurred, it may reveal unsuspected digitalis intoxication or serious poisoning, it sometimes provides help in the recognition of structural lesions in the heart or lungs, and it is often of great psychologic value in the examination of those who believe they have heart disease but whose hearts are actually normal.

* * *

Having mentioned briefly some of the possible contributions of the electrocardiographic method to medical practice, I must now concede that the present widespread employment of this method may actually be doing more harm than good, if one excludes its use in large hospitals. I make this statement quite soberly, and shall attempt briefly to indicate some of the reasons that I believe to be responsible for this anomalous and deplorable situation. My comments do not apply to most large hospitals, and not at all to those investigators, teachers and clinicians who have brought the knowledge of electrocardiography to its present high level and are utilizing it daily in their work.

It may simplify the discussion of this matter if one remembers that abnormalities in the electrocardiogram, broadly speaking, consist of those associated with the arrhythmias on the one hand, and those in the conduction intervals and the ventricular complexes on the other. In general, the serious errors of interpretation are confined to the latter and not to the diagnosis of arrhythmias, although even these are often unidentified or misnamed. It is the changes in the ventricular complex, including its terminal portion, that are mainly responsible for the gross misuse of a valuable diagnostic procedure. And the first statement I wish to make is that the average practitioner who undertakes to interpret electrocardiograms does not realize, or refuses to believe, that the changes in this part of the curve have no specific significance whatever, with the single exception of changes associated with myocardial infarction. To express it otherwise, the alterations in the conduction intervals or in the QRS complex and T wave on which he places emphasis in his diagnosis of coronary sclerosis or myocardial damage are not indications of any specific lesion in the heart, and may indeed be due to many causes other than heart disease. If this one truth could be accepted and remembered, most of the troubles with the method would end.

In the second place, the electrocardiogram almost never gives any helpful information about the functional state of the heart muscle, although thousands of physicians request such a record in the firm belief that the one who interprets the electrocardiogram will be able to determine exactly how much or how little activity the patient may be permitted. Actually, a very brief experience proves its uselessness in this respect and indicates what might be termed its positive and negative failure to portray the functional condition of the heart. For it has long been established beyond all possibility of doubt that the electrocardiogram may be perfectly normal in many patients whose hearts are seriously diseased, as in anginal or congestive heart failure. One may say with some confidence that the occurrence of acute myocardial infarction is about as clear evidence of coronary atherosclerosis as one can usually obtain during life, yet it is known that the conspicuous changes in the curve that often result from this condition may disappear wholly within a few months and thus leave the electrocardiogram normal. There is general agreement that a considerable percentage of patients who suffer from severe and unmistakable angina have normal curves, yet their hearts are abnormal. To indicate the failure of the electrocardiogram in the opposite direction, I need only state that certain well-marked alterations, often interpreted as indications of myocardial disease, may be found in the curves of persons who present no other signs of heart disease and who live for many years with no impairment of cardiac function and with no further change in the graphic records. Therefore, if it is known that a normal electrocardiogram may be obtained from a patient with advanced heart disease, and a distinctly altered curve from a person who has no clinical signs of heart disease, how can one possibly ask that the electrocardiogram reveal the functional condition of the myocardium? It is manifestly impossible, yet it is being asked scores of times every day.

A third misconception responsible for many errors is the fairly common belief that the electrocardiogram is an accurate quantitative indicator of the amount of digitalis active in the heart at a given moment. It is very difficult indeed to persuade some experienced clinicians that the record

will not tell precisely whether the patient needs a little more digitalis, a great deal more digitalis, or whether he has received a little too much and is on the verge of intoxication. As a matter of proved fact, the electrocardiogram will do no such thing. The curves from one patient may display marked changes preceding or accompanying the clinical signs of therapeutic effects, those from a second may show no changes (or very slight ones) when the patient is known to be fully digitalized, and those from a third may show only trivial alterations when there are clear signs of digitalis intoxication, as manifested by headache, nausea and vomiting. There are probably occasional cases in which the electrocardiograms give help concerning the administration of digitalis; it is quite certain that there are many in which they give none, and some in which they may actually be misleading if one is so foolish as to rely on these curves alone for guidance. The recent careful study of Geiger and his associates[2] has shown that depression of the ST segments, long regarded as an invariable effect of the administration of digitalis in therapeutic doses, occurred in less than half their digitalized patients. They also found — and this is perhaps of even greater consequence — that the onset and progression of such changes were not constant and were not quantitatively related to the amount of the drug that had been administered. Their study, which confirms the experience of many careful observers, indicates the impossibility of using the electrocardiogram as an index of the *degree* of digitalization.

There is another way in which these curves are often misused: some physicians still attempt to secure information about the prognosis from the electrocardiogram, although it has been shown again and again that this is usually impossible. In acute diphtheritic myocarditis, rapidly progressive changes in the ventricular complexes may justify the fear that death is imminent, and in occasional cases of acute myocardial infarction, similar progressive alterations may arouse the suspicion that the area of injury in the heart is larger and more serious than the clinical signs indicated. But even in these acute conditions, the graphic records must be interpreted in the light of the clinical evidence, while in chronic heart disease it is the clinical picture and laboratory data as a whole that must be used as the basis for estimating the prognosis.

Finally, and this is really but an amplification of my first criticism, misinterpretation of the electrocardiogram is responsible for a very large number of unjustified diagnoses of heart disease. The diagnosis of coronary disease is being made today almost as freely as that of vitamin deficiency, and with far less justification. But I doubt if many physicians other than those who specialize in this particular field realize the extent of this error or the tragic consequences that often follow. I am speaking with deliberate conservatism when I say that every week scores, if not hundreds, of normal healthy subjects are restricted in their activities and made to suffer needless apprehension because of diagnoses of heart disease based on minor changes in the electrocardiogram alone, in the absence of all supporting clinical evidence. This statement is amply warranted by my own experience, but I do not limit it to my practice or to the State of Connecticut. Like all others who specialize in this restricted field, I see many curves taken in various parts of the United States, brought to me for the most part by patients who have been condemned to a life of semi-invalidism solely because of changes in the electrocardiogram. In most cases, these changes consist of such unimportant alterations as minimal slurring of the R or S wave in one lead, low voltage of the ventricular deflections in one or two leads, and the presence of a large Q wave or inverted T wave in Lead 3. My friends and colleagues in other cities tell me that their experiences are exactly similar to mine. But perhaps the best indication that this misuse of the method is not confined to one part of the country is the fact that the American Heart Association has received letters from leaders in the cardiovascular field in the East, the South, the Midwest and the far West, asking if some board cannot be established for the sole purpose of certifying those physicians who are competent to interpret electrocardiograms. And why? Because they have been appalled at learning how many wholly untrained and incompetent physicians have purchased electrocardiographs and are using them daily in their offices, with serene confidence in their ability to distinguish between the important and the unimportant changes in the curves. In many cases, their erroneous diagnoses would be merely ridiculous if they did not so often lead to tragedy in the lives of their patients.

I believe one of the principal reasons why the electrocardiographic method is doing widespread harm today is that many physicians overemphasize its value and underestimate its limitations. They confidently expect it to accomplish much the same things that a surgeon expects of an exploratory laparotomy: that it will disclose the nature of the pathologic process, reveal its exact location and extent, and tell a great deal about the immediate treatment and prognosis.

They fail to realize that it is purely a laboratory procedure, comparable in many respects with a roentgenogram or a leukocyte count. As such, the electrocardiogram is to be interpreted merely as one finding among many, and in the light of all the clinical evidence and other laboratory tests. With the exception of the arrhythmias and some cases of acute myocardial infarction, it is reprehensible to attempt an interpretation of the electrocardiogram without knowing the clinical findings and the clinical diagnosis.

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RIGHT-UPPER-QUADRANT PAIN ON EFFORT: AN EARLY SYMPTOM OF FAILURE OF THE RIGHT VENTRICLE*

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PAINFUL congestion of the liver at rest, due to sudden stretching of its capsule, during right-sided heart failure is frequently encountered. This must be regarded, however, as a fairly advanced stage of failure, just as dyspnea at rest must be considered a distinctly advanced stage of left-sided heart failure. Even as the stage of constant pulmonary engorgement is frequently preceded by a period during which additional loads put on the left ventricle may result in temporary flooding of the lungs with blood, so, we believe, there is a period of weakness of the right ventricle when a greater burden will result in temporary congestion of the liver. This organ forms a capacious blood reservoir and serves as a pool for the general venous circulation in the early stage of right ventricular failure. Patients are not uncommonly seen in whom the liver is enlarged, but in whom there is little or no obvious venous distention or increase in venous pressure, both in the early stages of failure and during the course of recovery from advanced failure.

In the case that initiated our interest in this subject in 1938, the patient was a twenty-one-year-old student who had always been well — except for attacks of rheumatic fever at the ages of five, fourteen and fifteen years — until six weeks prior to admission, when he began to feel poorly and to tire easily. Two weeks later, he went to his home, at an elevation of 8000 feet, where he became miserable with palpitation, upper abdominal discomfort and anorexia. He was digitalized at that time, but without much benefit. The striking thing in the history on admission to the hospital was that the only specific complaint was a "tight feeling" in the right upper quadrant that came on regularly while he was walking up one flight of stairs at average speed and disappeared with rest. Physical examination revealed mitral stenosis, slight aortic regurgitation and auricular fibrillation. The only sign of heart failure at this time was a tender liver edge palpable two fingerbreadths below the costal margin. During the next two months, the patient developed progressive signs of right-sided heart failure, with distended neck veins and edema, and died with intractable congestive failure and rheumatic fever.

Following this experience, 3 more patients were encountered in whom the initial symptom of heart failure was pain in the right upper quadrant coming on with exertion and disappearing with rest. One patient was a forty-nine-year-old Negro whose chief complaint was right-upper-quadrant pain on effort. In this patient, an arrhythmia with tachycardia had been present for some years, and electrocardiography showed this to be auricular flutter with an auricular rate of 280 and an auriculoventricular block varying from 2:1 to 4:1, so that the ventricular rate averaged about 100 at rest. The electrocardiogram also showed inverted T waves in Leads 1 and 4, with a tendency toward right-axis deviation. The blood Hinton reaction was positive, but there were no heart murmurs, and x-ray examination showed no obvious abnormality of the aorta or significant cardiac enlargement. The pulmonic second sound was not accentuated. There was calcification at the roots of the upper and lower lobes of the right lung, with considerable emphysema and diaphragmatic adhesion on the right. Albuminuria was constant, and there was moderate anemia. The arterial blood pressure was 120 systolic, 80 diastolic. The venous pressure measured 200 mm. at first, but under treatment by digitalis, with reduction of the ven-

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tricular rate to 70 by an increase in the grade of block, it dropped to 96 mm. The liver was slightly enlarged. The patient was discharged in a much improved condition, with a diagnosis of auricular flutter and systemic venous congestion.

The etiology of this patient's heart failure remains obscure. At first, it was attributed to the effect of prolonged tachycardia, and this, in part at least, is doubtless the correct answer. He has since been followed for several months during which his failure has become progressively more marked, despite control of the ventricular rate by means of digitalis. The absence of signs pointing to pulmonary hypertension makes the usual causes of cor pulmonale unlikely. Recent x-ray evidence of increase of the heart shadow to the right, in addition to the electrocardiographic abnormalities, makes it probable that the chambers of the right side of the heart are involved primarily, whether from preponderant fatigue due to the tachycardia or from some unknown abnormality. Whatever the cause of the circulatory failure, the liver congestion was clear — at first, paroxysmal and, later, persistent.

Measurements of the circulation time and venous pressure were made on this patient before and after exercise. The exercise consisted in walking over the standard two-step stairs described by Master[1] at the rate of one trip in four seconds for a period of two minutes. It will be appreciated that this is only a very moderate grade of exercise, amounting to the ascent of two flights of stairs in a rather leisurely manner. Paraldehyde was the test substance used for measuring the circulation time from antecubital vein to pulmonary capillaries, the normal values by this method ranging from 3 to 9.5 seconds.[2] The venous pressure was measured by the direct method of Moritz and von Tabora.[3] The response of the venous pressure to exercise in normal persons is somewhat variable, depending on several factors — principally on whether the pressure is measured in an exercised or unexercised limb. According to McCrea, Eyster and Meek,[4] the pressure in the hand veins rises during and following exercises involving the use of the leg muscles. The rise may be several times the control value and may persist for over five minutes after cessation of the exercise. The extent of the rise depends in part on the severity of exercise and in part on the training of the subject. We have confirmed the finding of a rise in venous pressure following exercise in two normal persons. The finding of a comparable increase in the venous pressure after exercise in the patient with right-upper-quadrant pain and in another patient with a moderate degree of right ventricular failure was of considerable interest. The response of the venous pressure to exertion thus does not necessarily provide a crucial index of the competency of the right ventricle, although this unexpected finding deserves further study.

The behavior of the circulation time proved to be more helpful in separating patients with normal hearts from those with impaired myocardial function. Figure 1 shows the results of the

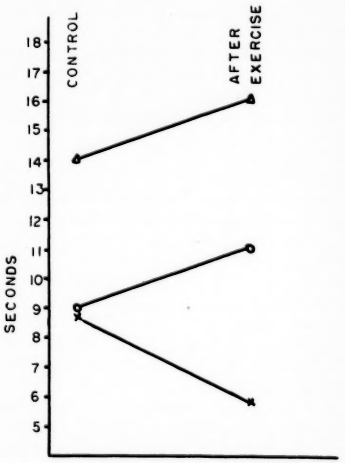

FIGURE 1. *Arm-to-Lung Circulation Time Before and Immediately Following Exercise.*

The upper figures (△——△) are those of a patient with moderately advanced right ventricular failure; the middle (o——o), those of a patient with paroxysmal right-upper-quadrant pain; and the lower (x——x), those of a normal control.

circulation-time tests in the patient with right-upper-quadrant pain cited above, in a patient with a moderate degree of right ventricular failure on the basis of long-standing mitral stenosis but with no history of abdominal pain, and in a normal subject. Since it has already been established that the circulation time regularly decreases in normal persons after exercise,[5, 6] the data on one normal control subject has been included to indicate that the method used is applicable to the data derived from other methods. It will be seen that in the normal subject the arm-to-lung time before exercise was a little less than 9 seconds, and that after exercise it had shortened to a little less than 6 seconds. The patient with clinically evident right-sided failure began with a definitely prolonged

arm-to-lung time,—that is, 14 seconds,— which was further prolonged to 16 seconds after exercise. The patient with the syndrome of right-upper-quadrant pain had a *normal circulation time to begin with, but after exercise it rose to a definitely abnormal level.* This abnormal reaction to exercise can be interpreted as meaning inability of the right ventricle to cope with the increase in venous return, with resultant slowing of the blood flow from the unexercised limb. Palpation of the patient's liver before and after exercise revealed no demonstrable change.

It has been our experience, as well as that of others, that the arm-to-lung time, although designed to measure the right-sided circulation, may be affected by congestive changes in the lung capillaries. Some patients with mitral stenosis therefore show prolongation of the paraldehyde time following exercise, even in the absence of significant heart failure. We were fortunate in encountering this patient with the signs of heart failure limited to the systemic circuit in whom changes of blood velocity, as measured by paraldehyde, could be evaluated in terms of efficiency of the right ventricle alone.

The following hint of the awareness of such a symptom as that under discussion was found in Vaquez's[7] book: "The functional disorders [of the liver] begin as subjective symptoms, slow digestion, belching of gas, bloating of the stomach, and a sensation of tension in the epigastrium and hypochondria which is aggravated by standing or walking." No other reference to this symptom on effort has come to our attention.

We have attempted to gather information on the incidence of this symptom and have found that in a small group of selected patients, numbering about 40, it was present in 18. The group selected for questioning were patients who either had definite right-sided failure at the time or were candidates for it by virtue of long-standing pulmonary hypertension secondary to mitral stenosis or, in a few cases, to left ventricular failure.

This direct questioning showed the reason why the symptom has not been emphasized heretofore and why it was mentioned in only 1 of 1000 case histories of congestive heart failure recently reviewed at this hospital. Only rarely was it of any severity, and it was frequently so overshadowed by subsequent developments as to lose significance in the patient's mind. The character of the pain was usually described as dull or aching in type, but also not infrequently as a feeling of tension or tenderness, and occasionally as quite sharp. The commonest precipitating factor was walking, but the pain was induced in 1 patient by paroxysms of coughing, and in 2 patients, although the pain was paroxysmal, no precipitating cause could be elicited (Table 1). It disappeared, as exertional dyspnea does, after a few moments of rest.

TABLE 1. *Characteristics of Intermittent Right-Upper-Quadrant Pain as an Early Symptom of Right Ventricular Failure.*

CASE NO.	AGE	SEX	CARDIAC LESION	TYPE OF PAIN	DEGREE OF PAIN	PRECIPITATING FACTOR
	yr.					
1	39	F	Rheumatic heart disease, with mitral stenosis	Aching	Mild	Exertion
2	38	M	Rheumatic heart disease, with mitral stenosis	Aching	Mild	Exertion
3	47	F	Rheumatic heart disease, with mitral stenosis	Aching	Moderate	Exertion
4	31	F	Rheumatic heart disease, with mitral stenosis	Sharp	Moderate	Cough
5	22	M	Rheumatic heart disease, with mitral stenosis	Aching	Mild	Exertion
6	38	F	Rheumatic heart disease, with mitral stenosis	Aching	Moderate	Exertion
7	29	F	Rheumatic heart disease, with mitral stenosis	Sharp	Moderate	Unknown
8	38	F	Rheumatic heart disease, with mitral stenosis	"Tired"	Mild	Exertion
9	21	M	Rheumatic heart disease, with mitral stenosis	Tension	Mild	Exertion
10	26	F	Rheumatic heart disease, with mitral stenosis	Sharp	Moderate	Exertion
11	33	M	Rheumatic heart disease, with mitral stenosis	Sharp	Moderate	Exertion
12	37	M	Rheumatic heart disease, with mitral stenosis	Aching	Mild	Exertion
13	49	M	Rheumatic heart disease, with mitral stenosis	Aching	Moderate	Exertion
14	61	M	Coronary heart disease	"Tender"	Severe	Unknown
15	67	M	Hypertension	Sharp	Moderate	Exertion
16	48	M	Aortic stenosis	Aching	Moderate	Exertion
17	55	M	Hypertension	Aching	Severe	Exertion
18	49	M	Long-standing auricular flutter	"Dragging"	Moderate	Exertion

The duration of the period of recurrence of this symptom seems to be short, usually a matter of two or three months, because, on the one hand, the patient regains some of his lost reserve or, on the other, failure progresses so as to stretch the liver capsule, when the probable mechanism of the symptom is lost. When 1 patient (Case 12) was questioned, he had only well-compensated mitral stenosis, but he was able to recall very clearly having gone through a period about four years previously when right-upper-quadrant pain on effort had been present. At that time, he was leading a very active life and doing moderately hard work. He consulted a physician and learned for the first time that he had rheumatic heart disease. He took his physician's advice to decrease his activity; the abdominal pain disappeared and had not recurred during the ensuing four years. Unfortunately, most of the other patients progressed to frank con-

gestive failure. Whether they might also have been spared advanced failure by the proper recognition and treatment of their early symptoms cannot be answered with confidence, but it seems likely that some, at least, might have been. Obviously, when a patient is precipitated into acute failure by, for example, auricular fibrillation with tachycardia, he may not pass through this stage of paroxysmal liver engorgement, but will at once present the more familiar picture of prolonged right-upper-quadrant pain and tenderness.

Summary and Conclusions

We have recently encountered 4 patients in whom right-upper-quadrant pain, precipitated by exertion and relieved by rest, has been the presenting symptom of early right-sided heart failure. It is evidently due to acute congestion of the liver and is comparable to dyspnea on effort in early left-sided heart failure.

Direct questioning of a group of 40 patients who already had clinically evident right-sided failure, or were likely candidates for it, revealed that the pain had been present at some time in about 45 per cent. It is a symptom to which the patient rarely attaches much significance, since it is usually of little severity and is overshadowed by more uncomfortable symptoms.

If the symptom is sought as diligently as the history of dyspnea on exercise, it may be found to be a fairly common and reliable warning of early weakness of the right ventricle.

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CONGENITAL HYDRONEPHROSIS IN THE LOWER HALF OF A DOUBLE KIDNEY*

Report of a Case

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MASSIVE congenital hydronephrosis filling half the abdomen and associated with reduplication of the ureter and renal pelvis has heretofore not been recorded. It is recognized that reduplication of the ureter wholly or in part and slight or moderate hydronephrosis are frequently encountered at routine cystoscopic examinations. However, a hydronephrosis filling half the abdomen from the diaphragm down into the bony pelvis is a rare condition. Cystic tumors large enough to displace the intestines to the opposite side of the abdomen present intriguing diagnostic problems. The following case was completely and accurately diagnosed preoperatively.

Case Report

G. D. (B 8902), a 5-year-old boy, was admitted to the Pediatric Service of the New Haven Hospital for study because of an abdominal mass discovered by a physician during a routine physical examination. The patient was said to have always had a protuberant abdomen, which had been growing steadily larger. There were no symptoms directly referable to the gastrointestinal or genitourinary tract, and the patient's general health had been excellent. Development and growth had been normal.

Examination revealed a well-developed and well-nourished boy who appeared to be in good health. The vital signs were normal, and the blood pressure was 100/58. Examination of the chest revealed no abnormalities. The abdomen was protuberant, with a marked rib flare, widening of the infrasternal angle and prominent, dilated superficial veins. The left side of the abdomen was larger than the right and was occupied by a smooth, cystic mass filling the left flank from the costal margin to the iliac crest and extending to well beyond the midline on the right. The remainder of the physical examination was negative.

Accessory clinical data were within normal limits. The blood showed a red-cell count of 3,870,000 with a hemoglobin of 12.5 gm., and a white-cell count of 8200 with a normal differential. The blood Kahn reaction was negative. Examination of the urine revealed a negative microscopic sediment and culture. The blood nonprotein nitrogen was 32 mg. per 100 cc. The tuberculin reaction was negative.

X-ray studies revealed elevation of the left leaf of the diaphragm and a large soft-tissue mass filling the left side of the abdomen. Gastrointestinal films showed the stomach to be displaced upward and to the right, with small and large intestinal loops pushed into the right half of the abdomen, the descending colon swinging in a curvilinear line over to the right (Fig. 1).

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Intravenous pyelograms showed normal-appearing internal kidney structures on the right (Fig. 2). On the left, there was a moderate hydronephrosis, with dilatation of all the calyxes visualized but with a suggestion of failure of filling of the lower portion of the kidney and its calyxes. The ureters were not outlined.

FIGURE 1. *Gastrointestinal Series, Showing Displacement of the Intestine to the Right Side of Abdomen.*

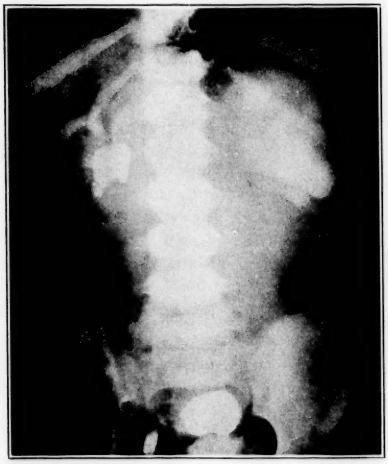

FIGURE 2. *Intravenous Pyelogram, Showing Hydronephrosis of the Upper Calyxes on the Left and Normal Internal Kidney Structure on the Right.*

Cystoscopy revealed a normal-appearing bladder and trigone. The ureteral orifices appeared normal, except that the left was not seen to function. The left ureteral catheter met an obstruction approximately 5 cm. above the ureterovesical junction; the obstruction was passed after slight manipulation, following which the catheter mounted readily but failed to drain. The pyeloureterogram on the right was normal (Figs. 3 and 4). The left ureter was

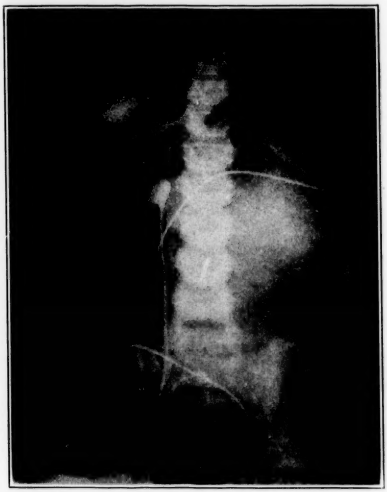

FIGURE 3. *Anteroposterior Film, following Retrograde Pyelography, Showing a Normal Right Ureter and Pelvis and Marked Displacement of the Left Ureter.*

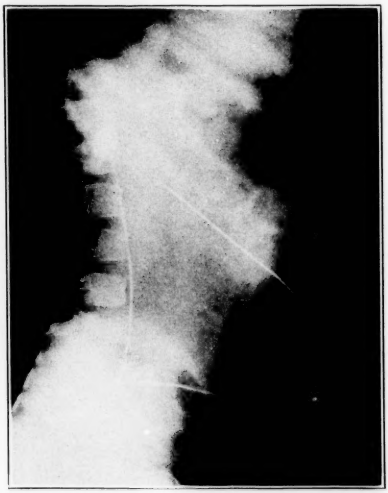

FIGURE 4. *Lateral Film, following Retrograde Pyelography, Showing Displacement of the Left Ureter beneath the Anterior Abdominal Wall.*

displaced to the right in the region of the 1st sacral segment, making a sharp bend at the iliac crest. It then

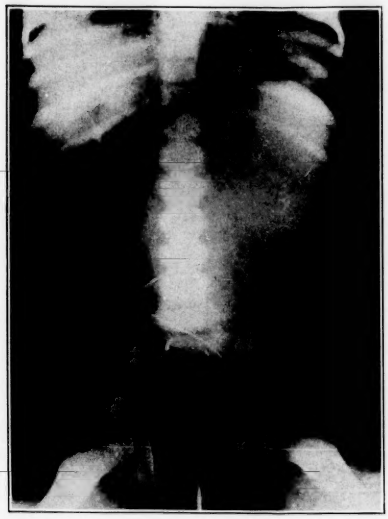

FIGURE 5. *Outline of the Hydronephrotic Lower Left Kidney following the Injection of Sodium Iodide.*

paralleled the right ureter to its upper third, where it crossed to run transversely toward the left renal area. displaced the ureter medially (Fig. 5); in the lateral view, this structure was seen anteriorly just beneath the abdominal wall.

Review of the intravenous and retrograde studies indi-

FIGURE 7. *X-Ray Film of the Specimen following Injection of Both Ureters with Sodium Iodide.*

cated a massive hydronephrosis in the lower segment of a double kidney, with complete destruction of functioning renal tissue in that pole, associated moderate hydro-

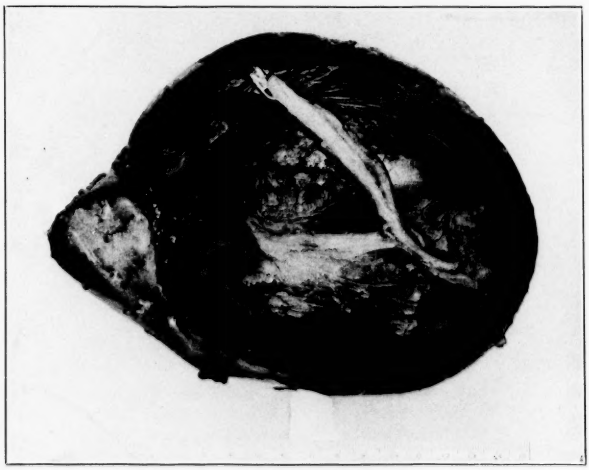

FIGURE 6. *Surgical Specimen.*

Successive injections of Skiodan, Diodrast and, finally, 12.5 per cent sodium iodide outlined a large, uniformly filled oval mass filling the left abdomen and flank, which nephrosis in the upper segment, and an incomplete reduplication of the ureter.

Accordingly, the patient was operated on through a left

Diabetes Insipidus

A dysfunction of the normal economy of the kidneys, rather than renal insufficiency, is a significant part of the pathogenesis of diabetes insipidus. With interruption or failure of consummation of the nervous impulses that normally traverse the supraopticohypophyseal tracts, the pituicytes of the neural hypophysis fail to elaborate a sufficient amount of antidiuretic hormone, and proper proportions of water and salt of the glomerular filtrate are not absorbed by the renal tubules. Polyuria, polydypsia and low urinary specific gravity (usually between 1.001 and 1.005) follow. The increased volume of urine excreted, contrary to casual impression, is not dependent on an increased rate of formation of glomerular filtrate. In fact, when glomerular filtration has been measured in patients with this malady, the value is on the low side of normal and not above normal.[9] The bulk of glomerular filtrate is reabsorbed by difference in osmotic pressure between blood and filtrate, and the antidiuretic hormone provides the stimulus for reabsorption of a smaller amount. The total reabsorption in a normal person is approximately 98 per cent. The excretion into the bladder of the remaining 2 per cent is sufficient to provide for a urine output of at least 2 liters. Reabsorption of glomerular filtrate in diabetes insipidus may be no more than 95 per cent complete. This reduction from the normal is sufficient to increase the daily urinary output to 10 liters. The percentile difference between reabsorption of fluid in a normal person and in a patient with diabetes insipidus, therefore, is not great.

Gout

The kidney has been held responsible for the pathogenesis and development of symptoms in patients with gout for several reasons. It is thought by some that a differential inability of the kidneys to excrete uric acid is the primary defect. I do not adhere to this assumption, for reasons presented elsewhere.[10] On the other hand, all students of the disease agree that renal insufficiency is a serious and frequent complication. This is thought to be caused by the burden imposed on the kidneys for the excretion of excessive quantities of uric acid. An increased excretion of uric acid might not be harmful to the kidneys if the solubility were similar to that of other salts, such as sodium chloride and potassium phosphate. The solubility of uric acid is not great, however, and the concentration in serum and glomerular filtrate in gouty patients is near the saturation point. When glomerular filtrate is concentrated in the convoluted tubules of the kidney, the maximum solubility for uric acid is exceeded and urate salts are precipitated. Persistent precipitation of urate in the tubular lumens eventually leads to renal insufficiency.[10] Patients who develop gouty symptoms in the early decades of life may succumb to renal insufficiency before the age of fifty.[11, 12] Patients who develop symptoms later in life may live a normal span of years. In addition to precipitation of urates in the tubules, urate sludge may appear in the kidney pelvis and may serve as a nidus for urate calculi. The urate stones are friable, and surgical intervention usually is unnecessary should they become lodged temporarily in the ureter.

The subject of renal impairment in gouty patients has been one of great interest to our laboratory. Many of the findings have been published recently in a survey of 22 patients with gout.[13] In this study, the function of the kidneys was investigated by routine clinical procedures, — that is, by ability to concentrate solids, excretion of phenolsulfonphthalein dye, and intravenous pyelography, — as well as by the clearance technics.[14] The patients were selected without regard to extent of disease or duration of symptoms of gouty arthritis. Their ages varied from twenty-eight to eighty-one. With the various procedures used, 18 patients showed some limitation of renal activity. The ability to concentrate solids was observed most frequently. More than two thirds of the group had a depressed inulin and creatinine clearance. Four had severe renal impairment, as shown by an increased concentration of nonprotein nitrogen in the serum. Death ensued in each of these patients in from two to four years after the azotemia was detected. Pathological studies confirmed the clinical findings of renal damage. There was no characteristic renal lesion, however, except for the deposits of sodium urate in the tubules, which were visible grossly and microscopically. Diffuse vascular changes were readily apparent, regardless of age. Evidence of glomerulonephritis and pyelonephritis was also noted. Treatment of the morphologic renal disturbance in gout is supportive rather than specific. An abundant urine volume with an alkaline reaction is desirable. This allays precipitation of urates in the tubular lumens.

Adrenal Insufficiency

Patients with Addison's disease during an adrenal crisis suffer from impending or complete renal shutdown, in addition to the more commonly appreciated metabolic dysfunctions. The ability to excrete phenolsulfonphthalein dye is decreased

markedly, urinary solids cannot be concentrated, and the concentration of nonprotein nitrogen in the serum may be 100 mg. per 100 cc. or greater.[15] More precise tests of renal function, that is, urea and creatinine clearance,[14] may be reduced 50 per cent or more.[16, 17] The mechanism is chiefly from reduction of glomerular filtration and follows hypotension, dehydration, decreased blood volume and hyperproteinemia. During treatment of acute adrenal insufficiency, evidence of severe renal impairment vanishes as the glomeruli resume filtration. Complete restoration of renal function is not achieved, however, and patients with Addison's disease whose symptoms are controlled by treatment with salt and hormones continue to show evidence of renal deterioration. This includes inability to concentrate solids maximally, albuminuria and cylindruria, as well as a depression in clearance of Diodrast and inulin.[18]

Patients with adrenal insufficiency secondary to pituitary hypofunction show similar changes. The impaired inulin and Diodrast clearances may be attributed to vasomotor unresponsiveness, decreased metabolic rate, nonspecific effect of a chronic disease and a lack of specific action by one or more adrenocortical hormones.

Hyperparathyroidism

Parathyroid adenoma or primary hyperparathyroidism is usually complicated by renal disease. The development of the pathologic changes in the kidney is well understood. With an increased concentration of parathormone in the body, serum calcium is increased, and urinary excretion of calcium and phosphate is augmented. Precipitation of calcium phosphate in the urinary tubules and appearance of calcium phosphate casts in the urine follow. The precipitation of calcium salt is enhanced by an alkaline urine. Prolonged deposition of calcium phosphate, with tubular and interstitial damage, — complicated frequently by infection and hydronephrosis, — leads to gross calcinosis in the renal pyramids and renal calculi, and subsequently to clinical renal failure. Albright et al.[19] emphasize the importance of excluding hyperparathyroidism in any patient with renal calculi. Removal of a single parathyroid adenoma or, in a few cases, of two or more diffusely so-called "hyperplastic" glands prevents further deposition of calcium phosphate but does not restore irreparable kidney damage.

Parathyroid hyperplasia secondary to renal disease has been called renal rickets as well as renal dwarfism. The term "rickets" has been applied because the bone changes have been confused with those seen in infantile rickets. The pathogenesis of renal rickets, however, is dissimilar to that of primary hyperparathyroidism or of infantile rickets. Diffuse hyperplasia of the parathyroid glands follows long-standing renal impairment.[20] The commonest cause of renal disease is a congenital anomaly of the genitourinary tract, although low-grade chronic nephritis, pyelonephritis, nephrosis and polycystic disease have been implicated in some cases. Even though renal damage begins early in life, the characteristic bone changes do not become evident until late childhood or adolescence. The blood pressure is usually normal throughout the disease.

Albuminuria, impaired concentrating ability, reduction of phenolsulfonphthalein excretion and nitrogen and phosphate retention define the amount of renal damage. At least three disturbances may be attributed to phosphate retention: increased output of phosphate in the gastrointestinal tract, impaired absorption of calcium from the gastrointestinal tract and hypocalcemia, and increased parathyroid activity and correction of hypocalcemia. These lead to abnormal bone formation and skeletal changes. At post-mortem examination, the kidneys are small, scarred and contracted — signs of a long-standing process. Histologically, they present diffuse sclerosis, obliteration of the glomeruli, and interstitial and intratubular calcification. Evidence of previous episodes of hypertensive nephrosclerosis is lacking. Attempts at restoration of renal function in renal rickets are fruitless. Considerable benefit to the bony structures can be achieved, however, by a high calcium intake and liberal amounts of a mixture of citric acid and sodium citrate.[21] Absorption of calcium is enhanced, and serum calcium is elevated, so that further stimulus to parathyroid activity is diminished. Normal deposition of calcium in bone is then possible.

Renal rickets with hypercalcemia is a rare syndrome, similar in many respects to typical renal rickets, but a different malady, nevertheless. A complete metabolic study of a patient with this syndrome has been reported from the Massachusetts General Hospital.[22] Renal tubular disease and inability to synthesize ammonia probably represent the underlying lesion. Increased amounts of calcium are excreted in the urine to replace ammonia, which is no longer formed. This leads to calcium depletion in the body, secondary hyperparathyroidism and decreased concentration of serum phosphate. It is therefore a low-phosphorus type of rickets as opposed to the usual high-phosphorus type.

Alkalosis

Temporary impairment of renal function may follow an acute episode of alkalosis, and morphologic deterioration of the kidneys is a complication of prolonged alkalosis. Cyclic vomiting in children and incomplete pyloric obstruction in adults give rise to alkalosis from excessive loss of hydrochloric acid in the gastric secretions. The prolonged ingestion of alkalies produces a similar effect on the hydrogen ion content of body fluids. The inherent danger of renal damage during the Sippy treatment of peptic ulcer was pointed out nearly twenty years ago by Hardt and Rivers.[23] The majority of patients who take soda bicarbonate do not develop clinical symptoms of alkalosis, although some degree of alkalinity of the blood must occur in all.[21] Intolerance for alkalies, rather than excessive ingestion, may be responsible for the development of renal symptoms. Kidneys with a low reserve become inadequate under the load imposed by need for increased excretion of base. Many of the patients who develop renal insufficiency during alkali ingestion are in the age groups that have a high incidence of chronic degenerative disease. It seems advisable to have reliable information regarding renal function in patients with pyloric obstruction and vomiting, as well as in those under consideration for prolonged alkali ingestion, and to appreciate the significance of the development of calcium casts and urinary calculi. Pathological studies of the kidneys of 6 patients who had received large amounts of alkalies during life have been reported by Cooke.[25] The tubules were the site of gross degeneration, lipoid changes and calcification, whereas the blood vessels, connective tissue and glomeruli showed little change.

Renal function has been studied by McCance and Widdowson[26] in alkalosis as well as after restoration of the acid-base balance of the blood. Clearances of inulin and creatinine were markedly depressed during alkalosis. Similar observations have been made in our laboratory. It is apparent that acute renal failure, which may develop as a critical episode in alkalosis from decrease in rate of formation of glomerular filtrate, is not necessarily associated with extensive anatomic damage.

Bilateral Cortical Necrosis of the Kidneys

This is a rare condition: less than 100 cases have been reported in the literature. It usually develops in women during or immediately following pregnancy. The child is almost always stillborn. A few cases of cortical necrosis following severe infections, such as dysentery, cholera, diphtheria and pneumonia, have been reported in both sexes. A negative past history for renal disturbance is usually obtained. Oliguria or anuria may be the first symptom that suggests renal damage. Elevation of the blood pressure, nausea, vomiting, edema and convulsions follow. If any urine is passed, it contains albumin, casts and blood cells. Few recoveries have been reported[27]; in fact, the correctness of the clinical diagnosis may be in doubt if the patient recovers. The kidneys are enlarged at post-mortem examination. The capsules strip easily and reveal a smooth surface, with mottled areas of gray yellow and dark red. The pathologic changes are confined to the cortex; the medulla is normal. Microscopically, the interlobular arteries are occluded, and the area supplied by each artery shows various degrees of necrosis in the tubules, glomeruli and afferent glomerular arterioles.[28] Observers agree that the pathological finding common to most cases is a vascular disease, but agreement is lacking whether endarteritis begins in the glomerular tufts and spreads centrally or whether necrotizing lesions of the interlobular arteries give rise to thrombi with secondary ischemia.[29]

Disseminated Lupus Erythematosus

Most patients with disseminated lupus show albuminuria, cylindruria and hematuria before death. These findings by themselves are not vital, since only a small percentage of patients with this malady suffer from renal insufficiency as the primary cause of death. It is significant, however, that once renal damage becomes evident, the prognosis is poor. Patients with this malady have been described who lived for several years without signs of renal damage, but died within a few months after this was evident.[30] A relatively frequent finding microscopically is the "wire-loop" appearance produced by hyaline thickening of the capillary walls.[31] There is also swelling of the glomerulus, cloudy swelling of the glomerular capsule, and irregularity and thickening of the basement membrane. According to Stickney and Keith[30]: "Clinically the duration of renal abnormality is much longer than the pathologic change would indicate: therefore, the disease either is not an ordinary glomerulonephritis or is of low virulence with slow progression. Possibly the changes are secondary to toxic processes and do not represent primary renal disease." The frequent occurrence of bacterial endocarditis, with renal emboli, is also responsible for anatomic changes.

Periarteritis Nodosa

The primary lesion in this malady is thought to be in the small arteries and arterioles. The vessels in the kidney are involved as part of the generalized disease. Renal findings usually noted

in periarteritis include albuminuria, cylindruria, hyposthenuria and red and white blood cells in the urinary sediment. The association of fever, polymyositis, polyneuritis, eosinophilia and renal disturbance justifies the consideration of this malady in differential diagnosis. Biopsy of a striated muscle is of great help in establishing a correct diagnosis.

Congenital Heart Disease

Patients with advanced symptoms of the tetralogy of Fallot[32] show albuminuria, poor concentration ability, azotemia and impaired pyelographic excretion of Diodrast. Structural changes in the kidneys may be attributed to one or more processes, which include venous congestion, anoxemia, acidosis, glomerular nephritis and subacute bacterial endocarditis with renal infarction. Routine clinical tests of renal function may be negative at earlier stages of the disease. A significant reduction in inulin and creatinine clearance,[14] however, may be demonstrated, as it was in several patients studied at the Massachusetts General Hospital in recent years.[33] This is an excellent example of the fallacy of concluding that the kidneys are normal if clinical tests only are employed.

In patients with coarctation of the aorta, all tests, including inulin clearance, may be negative except Diodrast clearance, which is depressed. The filtration fraction ($C_I \div C_D$)[14] may be increased to more than 25 per cent. Friedman and his associates[34] have assumed that reduction in afferent blood flow and renal ischemia is primary and is followed by elevation of blood pressure and efferent arteriolar spasm. Such an interpretation may prove eventually to be inadequate.

Nephrotoxic Agents

Hemoglobinuria, Transfusion Reactions and Blackwater Fever

The development of partial or complete suppression of urine following a transfusion is attributed to the agglutination of the donor's cells by the recipient's serum, with subsequent hemolysis and hemoglobinuria. The kidney lesion is primary in the tubules and is similar to that observed in blackwater fever.[35] It is caused by precipitation of acid hematin from the glomerular filtrate.[36] As the hydrogen ion concentration of the glomerular filtrate changes from alkaline to acid in the distal convoluted tubules, hematin is precipitated and rapidly plugs the tubular lumens. Pathological examination shows varying degrees of degeneration of the tubular epithelium, and leukocytes, debris and hemoglobin in the lumens. There may be cellular infiltration and edema of the interstitial spaces. The glomeruli are essentially normal. Opinion is divided concerning whether the pathologic changes result solely from mechanical plugging of the tubules or whether a toxic process is superimposed. Necrosis of the tubular cells and central focal necrosis of the liver argue in favor of a multiple pathogenesis.[37]

There is no specific treatment of anuria following hemoglobinuria. Appreciation of the factors that lead to a transfusion reaction may prevent it in some patients. Discovery of the Rh factor in blood grouping is a vital contribution in prophylaxis. This has recently been discussed by Hooker.[38] The early recognition of a transfusion reaction and cessation of further infusion of whole blood may be lifesaving. An intravenous infusion of an alkaline solution before the tubules have become plugged with acid hematin is theoretically sound, but it is of little benefit after the initial insult. From 100 to 200 cc. of sodium racemic lactate solution given intravenously will keep the tubular urine alkaline for several hours and prevent further precipitation of the degradation products of hemoglobin. Similar alkalinization or ingestion of soda bicarbonate by mouth may be useful in the prevention of transfusion anuria in patients who receive several transfusions of apparently compatible blood. Symptomatic treatment of anuria should not be neglected. Maintenance of the normal concentration of the constituents of the blood from day to day should be attempted. Recovery has been reported after anuria of from ten to twelve days' duration and an elevation of the nonprotein nitrogen to 200 mg. per 100 cc. Since the glomeruli suffer only secondarily, renal function may be restored once the tubules become patent. The acid-base balance of the blood should be maintained as effectively as possible until such restoration occurs. Decapsulation of the kidney is discussed below.

Crush Injuries

A new syndrome, an effect of the total-war principle of World War II, has been described in the British literature by Bywaters and others.[39, 40] It occurs among air-raid casualties who have been buried for several hours under great pressure on one or more extremities. The victim appears to be in fair clinical condition on rescue, and the involved structures show little more than edema and local anesthesia. Thereafter, a series of events rapidly develops that includes impairment of nutrition of the affected limb, signs of progressive renal insufficiency, oliguria, cylindruria, albuminuria and azotemia, circulatory collapse and intractable vomiting. The blood urea may rise to levels of 300 mg. per 100 cc., and the serum

potassium to levels of 7 milliequiv. per liter. Amputation of the affected limb does not stay the lethal processes, and the victim succumbs within a few days or a week. Advanced degenerative changes and brown-pigmented casts are seen in the convoluted tubules of the kidney at pathological examination. In place of hemoglobin from the red blood cells, the toxic agent is myohemoglobin,[41] liberated presumably from crushed muscles. This diffuses into the blood stream and is subsequently precipitated in the renal tubules. Janeway[42] has pointed out the desirability of early administration of alkaline fluids as a preventive measure. The problem deserves further study because of its importance in the treatment of civilian and military casualties. Furthermore, it is possible that a syndrome similar to this is an unrecognized industrial hazard following crush injuries in peacetime jobs.

Sulfonamide Derivatives

There is considerable variation among the several sulfonamides regarding the untoward effects on the kidney. Renal complications following the use of sulfanilamide are rare. Sulfathiazole and sulfapyridine are frequent offenders because of the tendency to form crystals in the lumens of the renal tubules. The sulfonamides are handled by the kidney as urea is handled, that is, they are present in the glomerular filtrate in the same concentration in which they exist in the plasma, and are reabsorbed in part by the tubules; the portion not reabsorbed is concentrated and excreted. Precipitation of crystals at the point of concentration in the tubules is unavoidable if adequate fluid is not available for solution. Sulfathiazole crystals appear in the urine more frequently than sulfapyridine crystals,[43] probably because the excretion of the former is more rapid and larger doses are needed for satisfactory blood levels. Plugging of the tubules with crystals may have dire consequences. Hematuria, anuria and retention of nitrogen products are direct effects. Equally alarming is the retention of the acetylated form of the sulfonamides. Since the acetylated forms are much more toxic than the free forms, the situation deteriorates rapidly, and a vicious circle is established. Caution seems indicated, therefore, in the use of sulfonamides in patients with recognized renal impairment. Because sulfonamide calculi are nonopaque to x-rays, this method of examination is not helpful in detecting them. Frequent examination of the blood for the concentration of the free and acetylated forms and examination of the urine for red cells and crystals must be relied on.

Microscopic examination of kidneys damaged by sulfapyridine shows extensive deposits of the acetylated form.[44] There is marked dilatation of the convoluted and collecting tubules and glomerular spaces by precipitated material. The tubules in the cortex appear more uniformly affected than those in the medulla. The prophylaxis and treatment of renal complications following sulfonamide therapy are important. A change of drug must be considered if the concentration of the acetylated form rises in the blood. An increased fluid exchange is helpful in redissolving precipitated material. Alkalinization of urine enhances the solubility of acetylsulfapyridine. The detection of hematuria and crystalluria is a caution signal but is not necessarily a signal for cessation of the sulfonamides.

Mercury Bichloride Poisoning

In acute mercury poisoning, the kidneys bear the principal insult, and many of the serious consequences can be traced to renal impairment. The tubules suffer most, and may be rendered functionless by the accumulation of large quantities of debris.[45] Complete cessation of renal function may persist for a week or longer, with an elevation of nonprotein nitrogen to levels over 200 mg. per 100 cc., and as in transfusion anuria, recovery eventually follows. Specific treatment of bichloride poisoning — that is, the administration of sodium formaldehyde sulfoxalate — may be a lifesaving measure in some patients, but the problem of controlling the chief cause of death remains unsolved. Sulfoxalate must be given orally or intravenously within a few minutes after the initial absorption of mercury to be effective. The end product of the reduction of mercuric chloride by sulfoxalate in vitro is about half as toxic as mercuric chloride.[46] The effectiveness of sulfoxalate is enhanced in an alkaline medium, and the use of alkalies is therefore desirable. Other measures that should be instituted at once to prevent additional absorption or reabsorption include lavage of the stomach and rectum and washing of the mouth.[47] Supportive treatment after the initial damage has been done is equally helpful. Unending efforts should be expended toward the treatment of shock and the maintenance of the acid-base balance of the blood, before, during and after anuria.[48] This includes the intravenous administration of whole blood, physiologic saline solution and alkaline solutions.[49]

Hepatorenal Syndrome

This designation is introduced with reluctance, since reliable observers have expressed doubt regarding the existence of a syndrome of essential

kidney and liver deterioration. The association of kidney disturbance with jaundice and liver disease is sufficiently common, however, to warrant a discussion, in the absence of general acceptance of a clinical syndrome. The association may be as subtle as the appearance of casts, blood cells and albumin in the urine of patients with surgical jaundice, or it may be as pronounced as in severe eclampsia with advanced renal failure and extensive and irreparable liver damage. An excellent bibliography of the literature prior to 1939 is given in a paper by Wilensky.[50]

There appears to be some confusion in the minds of physicians concerning the statement in textbooks that the liver is the site of formation of urea and that in liver disease a decreased content of urea in the blood may therefore be noted. Bollman, Mann and Magath[51] observed that the conversion of amino acids into urea occurs exclusively in the liver in the dog, and that urea production ceases following total extirpation of this organ. Protein metabolism does not cease, however, and amino acids accumulate in the blood if they are converted into urea. In the clinic, a counterpart of this situation is unique.[52] If only a small amount of functioning liver tissue remains, this is usually sufficient to make some urea, and complete obliteration of functioning liver tissue, with inability to form any urea, is a terminal event of short duration. An increase in concentration of urea in the blood, or no change, is therefore usual in liver disease. In a series of 180 cases of jaundice from medical and surgical causes, — including hepatitis, cirrhosis, and benign and malignant obstruction of the biliary passages, — Meyer, Popper and Steigmann[53] found a moderate increase of nonprotein nitrogen in the blood (40 to 70 mg. per 100 cc.) in nearly 40 per cent, and values greater than 70 mg. in 10 per cent. The mortality rate increased with a rise in the nonprotein-nitrogen level. Furthermore, a change in concentration was of prognostic significance, and an increase of nonprotein nitrogen in conditions amenable to surgery pointed toward the urgent need for surgical intervention.

The pathogenesis of renal insufficiency in liver disease is not yet established. If the hepatorenal syndrome is a clinical fact, kidney deterioration may be an integral process. On the other hand, azotemia may be extrarenal in origin, because of increased breakdown of tissue protein, dehydration and so forth. Whichever may be the correct explanation, azotemia suggests glomerular as well as tubular disturbance. Similar conclusions have been deduced from the study of urea and creatinine clearances[14] in patients with jaundice. These may be reduced from 20 to 75 per cent below normal.[53, 54] The pathologic picture suggests a degenerative, rather than an inflammatory, process that varies in degree from simple cloudy swelling to extensive necrosis of the tubular epithelium.[55] Glomeruli show proportionately less involvement.[56] Similar pathologic changes have been noted in animals following injection of bile salts (sodium dehydrocholate).[57] A major contribution has been made by Ayer[58] in the study of autopsy material from 18 infants who died of congenital atresia of the bile ducts. A constant microscopic finding in all cases was that of casts containing iron-free, brown pigment. They were present principally in the distal convoluted tubules and the collecting tubules of the cortex. Glomerular lesions were found in only 7 cases; 4 of these showed isolated and partly hyalinized bloodless loops, without epithelial proliferation. The severity of the renal changes was not related to the changes observed in the liver. A similarity between the renal lesions in transfusion reaction, bichloride poisoning and obstructive jaundice was stressed.

Decapsulation

The subject of decapsulation is of interest in a general discussion of nephrotoxic agents, since this form of therapy has been recommended in the treatment of anuria.[59] It is my opinion that nothing is to be gained by decapsulating anuric kidneys. If resumption of urine flow follows decapsulation, restoration would probably have followed conservative treatment. Since decapsulation is an extensive operation and patients with anuria are acutely ill, additional insults may be fatal and are best avoided. Recently, we had an unusual opportunity to study the effects of unilateral decapsulation in a young woman who developed anuria following a transfusion reaction. On the fourth day of anuria, the right kidney was decapsulated, and catheters were placed in both ureters. Resumption of urine flow began about twenty-four hours after operation. There was no difference, however, between the urine volume from the decapsulated kidney and that from the untouched organ. Six weeks later, after normal urine volume had been established for some time and the concentration of nonprotein nitrogen in the blood was almost normal, inulin and Diodrast clearances[14] were done. The ureters were catheterized again so that each kidney could be studied separately. A marked reduction in the rate of formation of glomerular filtrate and in renal blood flow was observed. More significant was the comparison of the two kidneys. No striking difference between the kidneys was noted in any of the functions measured. A

mathematical average of all functions, however, showed a slightly better performance by the untouched kidney in comparison with the decapsulated one. Our results are interpreted as implying that unilateral decapsulation in this patient had no beneficial action on renal function.

Effect of Posture

Orthostatic Hypertension

McCann and Romansky[60] reported clearance studies on 5 patients with hypertension associated with nephroptosis demonstrated by intravenous pyelography. The blood pressures in all patients were elevated when they were erect. Following a period of bed rest of from two to five days, the blood pressures were normal in all except 1 patient. The rate of formation of glomerular filtrate was unaltered by a change in position. On the other hand, the renal blood flow in the erect position was reduced by as much as 50 per cent, with a concomitant increase in filtration fraction. The treatment of only 3 patients is discussed. Two had a nephropexy performed, and 1 was given a ptosis belt. The therapeutic results were not particularly impressive.

Orthostatic Albuminuria

The current teaching that orthostatic albuminuria is a thoroughly benign mechanism is probably correct, although strict diagnostic criteria should be applied before acceptance of the diagnosis.[61, 62] Two important articles on this subject were written many years ago. Jehle[63] and Sonne[64] observed that orthostatic changes in renal activity occur usually in young, supple persons with a lordosis of the first and the second lumbar vertebra. This was evident from the following observations: when the patient was recumbent in bed, there was neither lordosis nor albuminuria; when the patient was recumbent in bed, with a pillow under the back, albuminuria appeared; standing produced lordosis and albuminuria; standing with the *left* foot on a stool corrected the lordosis, and the urine became clear. Sonne catheterized the ureters of 6 persons, and in no case was urine from the right ureter abnormal when the patient was erect; however, in 4 cases, the kidney on the left side became anuric, and in the remaining 2, the urine became albuminous. It was thought that the renal vein on the left was probably compressed by the vertebral column, since the vena cava lies to the right of the midline. These clinical observations have been confirmed by Chapman[65] and Rytand.[62]

Rytand studied intravenous pyelography by Diodrast, creatinine clearance and urinary constituents in 5 cases. The excretion of Diodrast and glomerular filtrate was significantly depressed in the erect position in 2, and similar to that in the supine position in 3. All showed casts, red blood cells and tubular epithelial cells in the erect position.

March Hemoglobinuria

An excellent review of this subject by Gilligan and Blumgart[66] has appeared recently. This is thought to be a rare condition, but the authors anticipate an increased incidence among men engaged in military activities if the possibilities of its development are appreciated. Hemoglobinuria and hemoglobinemia appear in young healthy men after strenuous marches or runs. In some subjects, lordosis appears to be a factor. The hemoglobinuria is functional in that renal damage cannot be demonstrated. The precise pathogenesis of the disturbance has not been determined, although the similarity of orthostatic albuminuria is apparent. The prognosis is excellent.

Gastrointestinal Hemorrhage

An increase of nonprotein nitrogen in the blood is a frequent accompaniment of massive bleeding into the intestine.[67] Several processes are responsible. Considerable quantities of hemoglobin remain in the stomach and intestines after severe hemorrhage, which, if assimilated, necessitates the excretion of excessive amounts of urea. The excess urea is cared for promptly, and azotemia is not observed if adequate fluids are available for excretion and if the kidneys are efficient. Usually, this is not realized because of interfering factors, such as shock, hypochloremia, dehydration and starvation, all of which are frequent sequelas of hemorrhage. Furthermore, patients suffering from internal bleeding are apt to be in middle or late life, ages that are accompanied by an increasingly high incidence of degenerative changes of the kidneys. Under conditions of stress, renal insufficiency is not unexpected.[68] Data contrary to this view have recently been presented by Stevens et al.,[69] who have concluded that azotemia following blood absorption is unrelated to renal impairment. It is apparent that further investigation is indicated before either conclusion is accepted as final.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 28061

PRESENTATION OF CASE

A seventy-two-year-old housewife was admitted to the hospital because of pain in the right lower quadrant of the abdomen.

The patient was quite well until the morning of the third day before entry, when she became aware of pain in the middle of her back in the lumbar region. Following lunch, at which she ate less than usual, there was flatulence, but no nausea, vomiting or pain. Some relief was experienced after the patient took "soda" and peppermint in water and lay down. Moderate amounts of gas were passed by mouth and by rectum. The patient slept comfortably that night. The next day, the pain in the back and the abdominal discomfort were replaced by pain in the right lower quadrant of the abdomen. At times, this pain was sharp and stabbing, although without radiation, and at other times it was a mere soreness. Moving about in bed was especially painful; walking was less so. The pain was aggravated by cough and by deep respirations. Bowel movements continued normal, and there were no urinary symptoms, nausea or vomiting. On the day before entry, the pain continued unchanged.

The past history and family histories were not of significance.

On admission, the patient had questionably jaundiced scleras. The heart was within normal size, with sounds of good quality and an apical systolic blow. There were basal "atelectatic" rales in the back, but the lungs were otherwise clear. There was exquisite tenderness and spasticity in the right upper and lower quadrants of the abdomen, with rebound tenderness referred to a point opposite the umbilicus. This point appeared to be the site of maximum tenderness. Peristalsis was active, with sounds of normal pitch.

The temperature was 100°F., the pulse 95, and the respirations 25. The blood pressure was 170 systolic, 70 diastolic.

Examination of the blood showed a white-cell count of 18,400 with 82 per cent polymorphonuclears. The urine was normal.

A roentgenogram of the abdomen showed considerable gas in the bowel, without dilatation. The kidney shadows appeared normal, and no stones were seen. The skeleton showed moderate osteoporosis.

An operation was performed immediately after admission.

DIFFERENTIAL DIAGNOSIS

DR. ALFRED KRANES: Before Dr. Hamlin starts his discussion, I may say that there was no question of jaundice in this patient.

DR. EDWARD HAMLIN, JR.: May we see the x-ray films?

DR. JAMES R. LINGLEY: Here are the loops of small bowel referred to in the record; they were not appreciably dilated. They appeared to be normal in size. Here is one loop, which is probably colon. I think that you can have that much gas in the small bowel just from abdominal pain.

DR. HAMLIN: We have, then, an elderly woman with a pathologic process in the abdomen; this apparently progressed to a fairly well-localized peritonitis in about twelve hours. The causes that might produce such a story and physical findings are, of course, numerous, and as always, one must first consider acute appendicitis. In an elderly woman, this would be a very good story, and the physical findings do not appear to be inconsistent with a diagnosis of acute appendicitis. The laboratory work all substantiates the diagnosis of peritonitis, or some acute episode in the abdomen, and I cannot exclude appendicitis. It is also possible, although much less probable, that she had acute cholecystitis. I was going to mention the questionable jaundice as possible substantiation of that diagnosis, but Dr. Kranes has ruled it out.

Numerous other processes should be mentioned as possibilities. Twisted ovarian cyst can produce a story and findings similar to these, with spasm masking the mass. Acute diverticulitis, possibly of the cecum, although rare, could produce such a story. However, one part of this history as printed that seems to me of more significance than anything else is the fact that the trouble began as pain in the middle of the back in the lumbar region. That should mean that the pain was initiated by a process, either retroperitoneal or in the mesentery, which would also be retroperitoneal. That brings up the possibility of pancreatitis, although this would be an atypical story indeed for pancreatitis, the localization of pain being particularly unusual. But it brings up the question, especially, in this age group, of mesenteric throm-

bosis. Mesenteric thrombosis can give a story of this duration. It may be argued that the normal bowel movements and the amount of gas passed by rectum should disturb one, but there is usually no mechanical obstruction of the bowel in mesenteric thrombosis, and if the area to be traversed is not too long, it is quite possible for the bowel contents and gas to pass down through a loop of intestine devoid of peristalsis, much as they would through a hose pipe. It is not unusual to find normal movements in cases of mesenteric thrombosis. It would be interesting to know whether guaiac tests were done on the stools, because at some time or other the patient should have passed blood.

DR. TRACY B. MALLORY: None are recorded.

DR. HAMLIN: It is difficult to determine whether the thrombosis was primarily in the arteries or in the veins. The length of the story, perhaps, is significant in that respect, and I shall make a primary diagnosis of acute venous thrombosis of the small bowel.

DR. LELAND S. MCKITTRICK: Dr. Hamlin has not made it quite clear to me how he has arrived at this very good diagnosis. He may well be right. It is a good diagnosis, but I wonder what makes him think so definitely that this is mesenteric thrombosis instead of the commoner conditions appearing in that region.

DR. HAMLIN: I should say that, to my mind, the main point is the pain in the back, and I tried to bring that out as significant. The location of the point of maximum tenderness, close to the umbilicus, is also of some importance. All the laboratory work and the story seem to be consistent with such a diagnosis, and I am at a loss to think of anything else that would quite fill the bill.

DR. MCKITTRICK: Pain in the back is not an uncommon symptom in strangulation obstruction of the bowel. I have seen one case of infarction of a large ovarian cyst in which pain in the back was the presenting symptom, and I just want to bring that out. It is interesting that Dr. Hamlin has taken pain in the back as the point that has made him think of interference with the mesenteric blood supply.

A PHYSICIAN: The pelvic examination is not mentioned.

DR. CLAUDE E. WELCH: It was entirely negative.

DR. KRANES: I believed that the patient had an acute inflammatory process in the right side of the abdomen, and that a possible interpretation of the back pain is an acute retrocecal appendicitis. I do not know how often that produces back pain, but a fair number of patients I have seen have had pain either in the flank or somewhat toward the back. Our preoperative diagnosis was acute appendicitis, with probable perforation and abscess formation, because of the exquisite tenderness and what felt like a mass on the right side. The mass was a little higher than one might expect with acute appendicitis. Dr. Welch saw her. He can tell what he thought about the situation.

DR. WELCH: The preoperative diagnosis was retrocecal appendiceal abscess,—although there were points that were not exactly consistent. For one thing, the operation was done at night, and something queer is always bound to turn up at that time. It was possible to palpate a mass under anesthesia. With a right-lower-quadrant (McBurney) incision, it was obvious that the appendix was not the cause. It was retrocecal and freed up easily. On further exploration, it was possible to feel a mass in the central portion of the abdomen. The incision was closed, and a paramedian incision revealed an infarcted twisted piece of omentum, about the size of a fist. In retrospect, this explains the pain in the back to which we had not paid adequate attention. It had a pedicle consisting of two blood vessels and nothing else. It had compressed the transverse colon but had not completely obstructed it. There may have been a good deal of pull on the mesentery of the bowel. The tip of the omentum had become anchored against the ascending colon a short distance above the cecum, and then proceeded on to infarction. The infarcted omentum was removed, and the postoperative course turned out to be uneventful.

CLINICAL DIAGNOSIS

Retrocecal appendiceal abscess.

DR. HAMLIN'S DIAGNOSIS

Mesenteric venous thrombosis.

ANATOMICAL DIAGNOSIS

Infarction of portion of the great omentum.

PATHOLOGICAL DISCUSSION

DR. MALLORY: The specimen which reached the laboratory was merely a segment of the great omentum, consisting of hemorrhagic, obviously necrotic, fat tissue.

CASE 28062

Presentation of Case

A twenty-year-old, single female clerk was admitted to the hospital because of lower abdominal pain of twelve hours' duration.

On the evening before admission, immediately after dinner, the patient developed severe, cramping abdominal pain, radiating from a point immediately above the symphysis pubis to the epigastrium. This pain was fairly continuous, but there were acute, colicky exacerbations. She was nauseated, vomited five or six times, and felt most comfortable lying on her left side, with the thighs flexed.

The patient had been well prior to the acute onset of this illness. Six months before admission, she experienced a similar attack, which was relieved in a few hours by aspirin. The menses were regular, at intervals of thirty to thirty-two days. The last period occurred three and a half weeks before admission. The family history was irrelevant.

On admission, the patient appeared somewhat dehydrated. The tongue was coated. The heart and lungs were normal. There was tenderness in the lower abdomen in the midline and slightly toward the right, most pronounced about 5 cm. above the symphysis. There was no spasm, but voluntary contracture was produced by pain. There was no rebound tenderness. The psoas sign was negative. On rectal examination, there was marked tenderness in both vaults, more so on the right than on the left. The uterus seemed in normal position, with slight tenderness on movement of the cervix. The vaginal introitus was virginal.

The temperature was 97°F., the pulse 100, and the respirations 20. The blood pressure was 122 systolic, 70 diastolic.

Examination of the blood showed a white-cell count of 14,000 with 95 per cent polymorphonuclears. The blood Hinton reaction was negative. The urine showed a + test for albumin.

On the first hospital day, an operation was performed.

Differential Diagnosis

Dr. S. P. Sarris: The problem in this case is essentially the differential diagnosis of an acute condition of the abdomen. Of course, in most cases, practically speaking, the surgeon is primarily interested in distinguishing between the surgical and the nonsurgical abdomen. This decision is made generally, I believe, on the physical signs. When it comes to the individual differential diagnosis of the various acute intra-abdominal conditions, one comes up against a much more difficult problem, since the symptoms and signs are common to so many acute surgical emergencies that only through the variation of minute details and the sequence of the history can one make an accurate differential diagnosis.

On the basis of real peritoneal tenderness, as shown by abdominal examination and confirmed by rectal and pelvic examination, I am going to exclude all medical and nonsurgical conditions. The first diagnosis that I shall consider is, of course, the commonest of the acute intra-abdominal surgical emergencies — acute appendicitis. It is true that one can almost never exclude acute appendicitis; however, I shall exclude that first and then continue. One of the reasons it cannot ordinarily be excluded is that appendicitis is so common that even its atypical forms are quite as common as any of the other rare intra-abdominal emergencies.

The first specific point for comment is the history of abdominal pain of twelve hours' duration. I believe that the time is important. Most patients with acute appendicitis come in within the second, third or fourth twelve-hour period. The fact that this patient came in the first twelve hours suggests that the pain was unusually severe. This point is confirmed later, when it is stated that the patient had severe, crampy abdominal pain. The pain in appendicitis is seldom very severe. It is more an "aggravating" type of pain. The onset, of course, is consistent with appendicitis, but I get the impression that this was a little more sudden than that in the average case of acute appendicitis. The character of the pain is consistent. The number of times that the patient vomited is rather against appendicitis. Usually, adults vomit either not at all or once or twice. This patient vomited five or six times within twelve hours. The previous episode of pain is consistent with appendicitis. Most patients give a history of a previous attack. When it comes to the physical signs, I am influenced more by those not in favor of the diagnosis of appendicitis than anything else. The tenderness seems to have been more diffuse than it is with an acute appendix. With a history of twelve hours' duration, that much tenderness by abdominal, pelvic and rectal examinations is more consistent with a perforated appendix, but very few perforate in the first twelve hours. Furthermore, the temperature of 97°F. is against the diagnosis of perforated appendix. Consequently, on the basis of the severity of the pain, the suddenness of onset, the frequency of vomiting and the

physical signs, I do not favor a diagnosis of appendicitis.

The next large group to consider is pelvic disorders. Fortunately, in this case, we have a virginal introitus. The case would be much more difficult if the situation were otherwise, because I should have a hard time excluding salpingitis. As it is, except for the rare blood-borne form of salpingitis, we can quite well exclude that diagnosis. The same argument holds for all conditions that are related to pregnancy. We must consider a ruptured corpus hemorrhagicum; the pain came on suddenly, and the physical signs are consistent with that diagnosis. However, there are two important things against the diagnosis, the chief one being the colicky nature of the pain. In ruptured corpus hemorrhagicum, the onset is acute, but the pain is constant and is more of a soreness from peritoneal irritation than acute pain. Also, the last period occurred three and a half weeks before entry, and a ruptured follicle usually occurs midway between periods.

I think that I can exclude all conditions associated with the upper abdomen. The physical signs and the location of the pain all point to the lower abdomen. Then we have certain conditions, such as acute appendix epiploica and acute Meckel's diverticulum, that I think are practically impossible to diagnose preoperatively. One may suspect them because of certain atypical features, particularly in anyone who has had an appendix removed and has an attack similar to appendicitis. In addition, this patient is too young for diverticulitis.

I might say a word about ulcerative colitis. We have had two cases that began with acute severe, crampy abdominal pain and lower abdominal tenderness without diarrhea. One case we believed to be appendicitis, and the correct diagnosis was made only four or five days after we had ochsnerized the patient for probable ruptured appendix.

Finally, we come down to what we might suspect is the diagnosis in this case, and at this point I should like to ask if any flat abdominal films were taken in this case.

DR. TRACY B. MALLORY: No.

DR. SARRIS: Apparently, those who saw this patient did not consider small-bowel obstruction. If they did, I assume that they would have taken a flat abdominal film. We do not have the common finding in the past history of small-bowel obstruction, — that is, a previous operation, — but 20 to 30 per cent of patients with small-bowel obstruction have not had a previous operation. There is no statement here concerning the bowels. Is there any about whether the patient's bowels had moved since the onset of pain, or about the passage of gas?

DR. MALLORY: I cannot find any.

DR. SARRIS: That would have helped, particularly if there had not been any bowel movement or passage of gas since the onset of the pain. In favor of the diagnosis of small-bowel obstruction are the sudden onset of the pain and its colicky nature. This patient apparently had continuous pain, with what is described as acute colicky exacerbations. There is no mention of the type of peristalsis this patient had — whether it was absent, present or increased. That is one of the most important physical signs of the diagnosis of acute small-bowel obstruction. So-called "obstructive peristalsis" is coincidental with acute exacerbations of crampy pain. The amount of vomiting is also more consistent with that diagnosis than with any other that I have mentioned.

DR. MALLORY: An additional note states that "peristalsis was present, but much diminished."

DR. SARRIS: Of course, I might have to change my diagnosis after that note. I should accept that finding if I were certain of the person who recorded it, because when one looks over the records of small-bowel obstruction, one of the features is the lack of consistency of that sign. One reason is, of course, that the examiner may not be looking for it. However, on second thought, diminished peristalsis could be consistent with small-bowel obstruction if circulatory involvement of the obstructed loop had occurred. On still further thought, I shall not let that single finding talk me out of what I think this patient had. The amount of tenderness is consistent with something more than small-bowel obstruction — namely, circulatory interference in the obstructed loop. Tenderness in simple small-bowel obstruction is usually minimal, and if it is present, it is usually localized to the obstructing point.

We can probably exclude the commonest single cause of small-bowel obstruction in a patient not operated on, namely, intussusception, which occurs almost always in infants. The other causes are fairly equally distributed between congenital bands, volvulus, Meckel's diverticulum and foreign bodies, such as gallstones. I think that in this case we can probably exclude gallstones, and that leaves us with congenital bands and a Meckel's diverticulum. Finally, I wish to come back to the question of an acute Meckel's diverticulum, which as I said before falls in a group on which one cannot make an accurate diagnosis preoperatively. Furthermore, I have seen only one case of simple acute diverticulitis. Usually, this disease

is associated with massive gastrointestinal bleeding or small-bowel obstruction. Accordingly, unless this patient had small-bowel obstruction associated with a Meckel's diverticulum, I do not believe that I can make such a diagnosis.

My first diagnosis in this case is acute small-bowel obstruction, with probable, at least early, circulatory interference; my second diagnosis is acute appendicitis. Incidentally, I almost overlooked the possibility of a twisted ovarian cyst, which is, of course, fairly common at this age. However, I exclude it because of the character of the pain, and the fact that the examiners could not feel a mass. If they could feel the virginal uterus, they should have been able to feel an ovarian cyst.

A Physician: Is not the pain just over the symphysis low for small-bowel pain?

Dr. Sarris: I am glad you brought that up. Most textbooks state that the pain in small-bowel obstruction is at or above the umbilicus. In the series of cases that I looked up with Dr. McKittrick, we found one third or more with pain in the lower abdomen.

One other point regarding the diagnosis of acute appendicitis — we are not told that the pain had shifted. If a careful history is taken, one can elicit a shifting pain in acute appendicitis in as high as 85 or 90 per cent of the cases. I do not mean necessarily a shifting from the epigastrium to the right lower quadrant, but a difference in the character of the pain, vague and crampy, followed by tenderness — soreness more than pain. That is absent in this history, unless it was a case of acute appendicitis before localization. I remember one such case we thought had small-bowel obstruction, and while the patient was getting x-rayed, over a period of two or three hours, the pain changed. When he came back to the Emergency Ward, he said that the pain had stopped, and he then could feel soreness on the right side. He had acute appendicitis. It is possible that this patient came in earlier than the average case and before localization.

Clinical Diagnosis

Acute appendicitis.

Dr. Sarris's Diagnosis

Acute small-bowel obstruction, due to either a congenital band or a Meckel's diverticulum.

Anatomical Diagnoses

Obstruction of small bowel.
Congenital adhesive band.
Gangrene of Meckel's diverticulum.

Pathological Discussion

Dr. Mallory: The preoperative diagnosis on the wards was acute appendicitis. At operation, an infarcted piece of intestine, looking like small bowel on first examination was seen and on further exploration turned out to be the end of a Meckel's diverticulum. There was a congenital band, which contained a rather large artery, running across to the base of the Meckel's diverticulum. Behind it, three coils of small bowel had been caught so that there was intestinal obstruction. The Meckel's diverticulum was gangrenous but not perforated.

Dr. Sarris: How about the intestine itself?

Dr. Mallory: That was not infarcted. The appendix was removed and was normal.

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THE JOURNAL does not hold itself responsible for statements made by any contributor.

COMMUNICATIONS should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston, Massachusetts.

UNITED WAR FUND CAMPAIGN

THE quota of the Physicians' Group for the United War Fund Campaign is $37,415. To raise this sum, seven hundred and fifty physicians must contribute $50 each. Obviously, since many are unable to afford such a donation, others are expected to make up the difference.

At the close of the first week of the drive, January 29, approximately 25 per cent of the total quota had been received. This amount represents fair progress toward the realization of the goal, judged by the number of pledges made. However, many have given much more than $50 per person — indeed, two physicians gave $1000 each. And the sum pledged seems far less encouraging when compared with the donations received in the campaign as a whole: $3,625,000, or nearly 50 per cent of the $7,600,000 goal.

Thus, it is apparent that only through great generosity can the quota of the Physicians' Group be raised. Previous drives have shown that the most substantial gifts have been received from those who have budgeted their pledges. Physicians are urged to give their fair share — in periodic payments, if necessary. They will thereby show their response to the patriotic and humanitarian appeal of this drive for victory on the home front and help for those who need it most.

RECOMMENDATIONS TO MEDICAL STUDENTS AND PHYSICIANS CONCERNING THE NATIONAL EMERGENCY

THE following recommendations to medical students and physicians deserve detailed consideration by all concerned. The efficient utilization of the existing facilities offered by the medical profession and a guarantee of the continued functioning of all sources of medical education are so important in the present national emergency that the procedures advised in this statement by the Procurement and Assignment Service should not go unheeded.

MEDICAL STUDENTS

All students holding letters of acceptance from the deans for admission to medical colleges and freshmen and sophomores of good academic standing in medical colleges should present letters or have letters presented for them by their deans to their local boards of the Selective Service System. This step is necessary in order to be considered for deferment in Class II-A as a medical student. If local boards classify such students in Class I-A, they should immediately notify their deans and, if necessary, exercise their rights of appeal to their local boards of appeal. If, after exhausting such rights of appeal, further consideration is necessary, request for further appeal may be made to the state director and, if necessary, to the national director of the Selective Service System. These officers have the power to take appeals to the President.

Those junior and senior students who are disqualified physically for commissions are to be recommended for deferment to local boards by their deans. These students should enroll with the Procurement and Assignment Service for other assignment.

All junior and senior students in good standing in medical schools, who have not done so, should apply immediately for commission in the Army or the Navy. This commission is in the grade of Second Lieutenant, Medical Administrative Corps, Army of the United States, or Ensign H-V (P), United States Naval Reserve, the choice as to Army or Navy being entirely voluntary. Applications for commission in the Army should be made to the corps-area surgeon of the corps area in which the applicant resides, and applications for commission in the Navy should be made to the commandant of the naval district in which the applicant resides. A medical R. O. T. C. student should continue as before, with a view of obtaining a commission as First Lieutenant, Medical Corps, on graduation. Students who hold commissions, while the commissions are in force, come under the jurisdiction of the Army and Navy authorities and are not subject to induction under the Selective Service Act. The Army and Navy authorities will defer calling these officers to active duty until they have completed their medical education and at least twelve months of internship.

RECENT GRADUATES

On successful completion of the medical-college course, every person holding a commission as Second Lieutenant, Medical Administrative Corps, Army of the United States, should make immediate application to the Adjutant General, United States Army, Washington, D. C., for appointment as First Lieutenant, Medical Corps, Army of the United States. Every person holding a commission as Ensign H-V (P), United States Naval Reserve, should make immediate application to the commandant of his naval district for commission as Lieutenant (JG), Medical Corps, United States Naval Reserve. If appointment is desired as Lieutenant (JG), Medical Corps, United States Navy, application should be made to the Bureau of Medicine and Surgery, Navy Department, Washington, D. C.

TWELVE-MONTH INTERNS

All interns should apply for a commission as First Lieutenant, Medical Corps, Army of the United States, or as Lieutenant (JG), United States Navy or Naval Reserve. On completion of twelve months of internship, except in rare cases in which the necessity of continuation as a member of the staff or as a resident can be defended by the institution, all who are physically fit may be required to enter military service. Those commissioned may then expect to enter military service in their professional capacity as medical officers; those who failed to apply for commission are liable for military service under the Selective Service Act.

HOSPITAL-STAFF MEMBERS

Interns with more than twelve months of internship, assistant residents, fellows, residents, junior staff members and staff members under the age of forty-five, fall within the provisions of the Selective Service Act, which stipulate that all men between the ages of twenty and forty-five are liable for military service. All such men holding Army commissions are subject to call at any time, and only temporary deferment is possible, on approval of the application made by the institution to the Adjutant General of the United States Army certifying that the individual is temporarily indispensable. All such men holding Naval Reserve commissions are subject to call at any time at the discretion of the Secretary of the Navy. Temporary deferments may be granted only on approval of applications made to the Surgeon General of the Navy.

All men in this category who do not hold commissions should enroll with the Procurement and Assignment Service. The Procurement and Assignment Service under the executive order of the President is charged with the proper distribution of medical personnel for military, governmental, industrial and civil agencies of the entire country. All those so enrolled whose services have not been established as essential in their present capacities will be certified as available to the Army, Navy or governmental, industrial or civil agencies requiring their services for the duration of the war.

PHYSICIANS UNDER FORTY-FIVE

All male physicians in this category are liable for military service, and those who do not hold commissions are subject to induction under the Selective Service Act. In order that their service may be utilized in a professional capacity as medical officers, they should be made available for service when needed. Wherever possible, their present positions in civil life should be filled, or provisions made for filling their positions, by physicians over forty-five, by physicians under forty-five who are physically disqualified for military service, by women physicians and by instructors and those engaged in research who do not possess an M.D. degree whose utilization would make available a physician for military service.

Every physician in this age group will be asked to enroll at an early date with the Procurement and Assignment Service. He will be certified for a position commensurate with his professional training and experience as requisitions are placed with the Procurement and Assignment Service by military, governmental, industrial and civil agencies requiring the assistance of those who must be dislocated for the duration of the national emergency.

PHYSICIANS OVER FORTY-FIVE

All physicians over forty-five will be asked to enroll with the Procurement and Assignment Service at an early date. Those who are essential in their present capacities will be retained and those who are available for assignment to military, governmental, industrial and civil agencies may be asked by the Procurement and Assignment Service to serve those agencies.

The maximal age for original appointment in the Army of the United States is fifty-five. The maximal age for original appointment in the Naval Reserve is fifty.

All inquiries concerning the Procurement and Assignment Service should be sent to the Executive Officer, 5654 Social Security Building, 4th Street and Independence Avenue, S.W., Washington, D. C., and not to individual members of the Directing Board or of committees thereof.

"SPEEDING PRODUCTION OF PHYSICIANS"

UNDER this title, the January 17 issue of the *Journal of the American Medical Association* carries an editorial in which doubt is cast on the wisdom of the shortened schedule adopted by the medical schools of Boston and commented on in the January 29 issue of the *New England Journal of Medicine*. Briefly, the plan provides that the three months heretofore passed in the long summer vacation be eliminated, thus allowing the entrance and the graduation of a class every nine, instead of every twelve, months. After quoting "one leading educator" to the effect that the scheme had not received mature consideration, the editorial goes on to list five difficulties that have arisen. These problems and somewhat different interpretations of them are as follows:

(1) *As an emergency measure, the immediate gain is slight.* The gain may not be great, but it is real and predictable.

(2) *With the elimination of summer vacations, some students will be forced to discontinue their medical education, owing to their inability to earn the necessary funds.* If the Government, particularly in view of its 1942 model of economy, cannot recognize the investment value of a medical student, when physicians are needed so badly, it had better revamp the Selective Service System. Actually, the Government does appreciate the value not only of the medical student but also of the premedical student, and medical schools and colleges could make no better investment than to stake their needy students at a low rate of interest.

(3) *Prescribed R.O.T.C. field training must be discontinued.* If the R.O.T.C. cannot accommodate themselves to anything other than "business as usual," they are overlooking their own interests and responsibilities in the urgency of the moment.

(4) *The technical and teaching staffs must be greatly increased or must devote far more time to their work.* Each medical school must, of course, decide what it is able to do. So far, there has been little, if any, complaint of overwork from the staffs of schools that have adopted the shortened schedule. Furthermore, the medical schools that remain on the old schedule will be a convenience to those college students who cannot complete their premedical requirements until September.

(5) *Unless colleges and hospitals have their curriculums suitably integrated with those of medical schools, the students will lose three to six months waiting for admission as medical students or as interns.* Such a condition has always existed, and there seems to be little necessity of adding another three months each year in the summer.

That professional standards must be lessened is undeniable and inevitable. They have already been lowered by the reduction of internships to one year, and by the renunciation of the somewhat grandiose thought, recently developed, that it takes six or eight years of hospital residence to make a surgeon. To protect the premedical student, it may be necessary to trim medical-school requirements. Physicians with teaching positions will have more work to do, in addition to that already demanded by plans for air-raid protection and civilian defense. Only time can be saved now. Yesterday's leisurely standards must be abandoned. Perhaps the emergency is a little more real and vivid to those who live along the seacoasts than to those in Chicago.

MEDICAL EPONYM

MILROY'S DISEASE

William Forsyth Milroy (b. 1855), professor of clinical medicine and hygiene in the Omaha (Nebraska) Medical College, read a paper entitled "An Undescribed Variety of Hereditary Oedema"

before the Society of the Alumni of Charity Hospital on June 1, 1892; this was published in the *New York Medical Journal* (**56:** 505-508, 1892). He described the condition as follows:

> On August 20, 1891, Mr. H. presented himself for examination for life insurance. . . . The applicant called my attention to his lower extremities. I found a condition of oedema involving the feet and extending up the legs to the knees. . . . Upon inspection, the leg presented a slightly rosy hue. . . . Scattered thickly over this base were white spots about the size of a pea. . . . Mr. H. stated that this oedematous enlargement had existed from birth. . . that this enlargement of the extremities was a family characteristic which he had inherited from his mother. Fortunately for the purpose of this study . . . in 1883 a member of the family published a neat volume, giving the family history in America for a period of two hundred and fifty years. . . . The peculiarity now under discussion seems to have entered the family by marriage about 1768. With the aid of this volume and the assistance of members of the family still living, I am able to offer the facts which I present. . . .
>
> 1. So far as known, in every case, with two exceptions only, the oedema was present at birth.
>
> 2. The location of the oedema has in every case been limited to one or both lower extremities.
>
> 3. The presence of the oedema is persistent, never having been known to disappear, temporarily or permanently, except in one instance.
>
> 4. It has never been attended by constitutional symptoms, barring the two possible cases in which its first appearance was subsequent to birth.
>
> From these considerations it seems evident that the case under discussion is not one of angeio-neurotic oedema. . . . Whether or not the case is one of nervous edema, it is offered that, with others, sufficient material may be accumulated to render possible study of these unusual forms of oedema.
>
> R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

COMMITTEE ON MATERNAL WELFARE

Causes of Maternal Death in Massachusetts during 1940 (*continued*)

Rupture of Uterus

One case in which autopsy was performed and a diagnosis of ruptured uterus established was an example of very poor obstetrics. An attempt to deliver the patient at home had been made by her physician, and when she arrived in the hospital, her pulse was 130 and she was in shock. The physician had diagnosed a prolapsed arm, and a version had been unsuccessful. A Braxton-Hicks maneuver was performed shortly after entry, and delivery was completed several hours later, with craniotomy of the after-coming head. The rupture of the uterus was not diagnosed; had it been appreciated, laparotomy with probable hysterectomy might well have saved this patient's life.

Another patient with a ruptured uterus died on the table, undelivered. Forceps delivery had been attempted, and version had been tried unsuccessfully before death. Post-mortem examination revealed a rupture of the lower segment. Such a death is inexcusable.

Another case of ruptured uterus occurred in association with a partial placenta previa. Version was done, and the patient, while on the table, went into profound collapse and died six hours later. Although no autopsy was performed, this was undoubtedly a case of ruptured uterus.

The next case of ruptured uterus was associated with an abnormal fetus and operative delivery. To deliver the fetus, craniotomy was necessary. During the process, the patient rapidly failed and died one hour after she was delivered. No x-ray film had been taken in this case to determine the size of the fetus, and this death can be attributed only to poor obstetrics.

One case was associated with a long-drawn-out, poorly handled labor. Three ½-ampule doses of pituitary extract were given during labor. The patient died very soon after a difficult forceps delivery. There is no excuse for this death.

In the next case, which was associated with a previous cesarean section, the patient went into spontanous labor, and the uterus ruptured before she was delivered. Autopsy confirmed the diagnosis.

In the second case with a previous cesarean section, the patient died of a rupture of the uterus during labor. This case was poorly handled throughout.

Another patient, who had had five previous cesarean sections, was in the hospital thirty-six hours before operation. Low cervical section was performed, and the patient's condition is said to have been excellent. Six hours later, she went into profound shock and died after four hours. Autopsy showed that the rupture occurred in conjunction with an old scar. The incision of the present operation was intact. This death is hard to explain.

Of these 8 cases,* 3 were associated with previous cesarean section. This fact emphasizes the seriousness of the initial section, and certainly proves that scars of previous cesarean sections may

*Further study of the cases of maternal death in 1940 has necessitated a change in the classification of the cause of death in several. Hence, the total numbers of cases in many of the groups do not agree with those given in the table in the January 8 issue of the *Journal*. A corrected table will be published in a subsequent issue.

rupture. The last case mentioned, in which an autopsy showed the rupture quite separate from the new incision, is most illuminating as well as very unusual. Those cases of ruptured uterus associated with operating are all examples of very poor obstetrics. These can never be eliminated until obstetrics is more carefully supervised in all obstetric hospitals.

Ectopic Pregnancy

Six cases of ectopic pregnancy, 5 tubal and 1 abdominal, resulted fatally.

One patient with tubal pregnancy was operated on five hours after the onset of acute symptoms. Much free blood was found in the abdomen. This patient died of peritonitis, five days after operation.

Another case, which was viewed by the medical examiner and in which no autopsy was performed, was undoubtedly one of exsanguination due to rupture in tubal pregnancy. The patient was not operated on, and the immediate history was not available. She was dead when she reached the hospital.

One patient died during operation. It is quite possible that spinal anesthesia was the primary cause of death, since the operation was begun within a short time of the onset of symptoms, when the patient was said to have been in good condition.

Another patient reached the hospital with a blood pressure of only sixty. This patient died without operation. No transfusion was given.

One patient died, not of the tubal pregnancy but of some pulmonary complication, five weeks subsequently.

There was 1 case of abdominal pregnancy, discovered at autopsy. The diagnosis was entirely missed, and the death was surely avoidable.

Surgical Shock

In 4 cases, the patients died very shortly after cesarean section, and hemorrhage did not seem to be the prime cause of death; hence, they are classified as due to surgical shock. In 1 of these cases, attempts at forceps delivery and version had failed, and in 1 case, a classic cesarean section was performed by a general surgeon. There is nothing to add about the other 2 cases, except that they, too, were badly handled.

One patient was operated on for a retained placenta previa. The history makes little mention of hemorrhage, so that death must be attributed to shock. Autopsy revealed that the uterus was not ruptured, and that a placenta accreta existed. The diagnosis of placenta accreta, of course, should have been made before death. Had this diagnosis been made, transfusion and hysterectomy might well have prevented this death.

Surgical Diseases

Acute appendicitis. Three cases of acute appendicitis resulted fatally.

One patient was between five and six months pregnant. The appendix had ruptured before the operation, and the patient died of general peritonitis five days later.

Another patient — about six months pregnant — who was operated on for acute appendicitis aborted eight days after operation. Her condition grew worse, and she died seventeen days after operation.

Another case of acute appendicitis occurred in a patient who was six months pregnant. At operation, the appendix was gangrenous; free pus was found in the abdomen, and the patient died on the fifth day after operation. She aborted spontaneously just before death.

It is very difficult to state that any of these deaths might have been prevented, but if they were under a physician's care from the onset and not diagnosed until after peritonitis developed, the physician certainly should be considered culpable.

Intestinal obstruction. One patient was operated on for acute intestinal obstruction, which autopsy proved to be due to adhesions that had followed a gastroenterostomy. This patient was about six months pregnant, and as a last resort, four days before death, delivery was induced. This procedure certainly cannot, in any way, be condoned.

Another patient, who was four and a half months pregnant, died of intestinal obstruction. At operation, it was found that the obstruction was due to adhesions from a previous operation.

One patient, when about seven months pregnant, entered the hospital with a history of having vomited for a few days, with a great deal of abdominal pain. An operation for intestinal obstruction was performed, and at the same time, cesarean section was done. The patient failed, and died of complete intestinal obstruction. If intestinal obstruction demands operation, the uterus should be left alone.

In another case, the patient was about four and a half months pregnant and was under observation at the hospital for five days before the diagnosis of intestinal obstruction was made. This patient aborted shortly before death. Intestinal obstruction was due to adhesions from previous

operations. The operation to relieve the obstruction was undertaken too late to be of any value.

It should be axiomatic, in all cases of intestinal obstruction that occur during pregnancy, that the pregnancy be disregarded and that the obstruction be treated; the sooner the operation is performed, the greater the likelihood of recovery. It certainly is a reflection on the medical profession of Massachusetts to allow a patient to remain in the hospital five days before diagnosis and operation, as was true in one of these cases.

DEATHS

DOWNEY — HUGH J. DOWNEY, M.D., of Pittsfield, died January 31. He was in his sixtieth year.

Born in Adams, Dr. Downey received his degree from Chicago College of Medicine and Surgery in 1912. He was a former secretary and president of the Berkshire District Medical Society. He was a member of the Massachusetts Medical Society and the American Medical Association.

His widow, a son and a daughter survive him.

POTTER — A. CARLETON POTTER, M.D., of Cambridge, died January 28. He was in his sixty-ninth year.

A native of Cambridge, Dr. Potter received his degree from Harvard Medical School in 1899. At the time of his death he was president of the New England Society of Physical Medicine, and was a fellow of the Massachusetts Medical Society and the American Medical Association.

His widow, a daughter and a sister survive him.

WEISS — SOMA WEISS, M.D., of Boston, died January 31. He was in his forty-fourth year.

Born in Hungary, Dr. Weiss received his degree from Cornell University Medical College in 1923. He was assistant at the Thorndike Memorial Laboratory from 1925 to 1929, assistant in medicine at Harvard Medical School from 1926 to 1927, instructor from 1927 to 1929, assistant professor from 1929 to 1932 and associate professor from 1932 to 1939; he was appointed Hersey Professor of the Theory and Practice of Physic in 1939. He was assistant director of the Thorndike Memorial Laboratory from 1930 to 1932, director of the Second and Fourth Medical Services (Harvard) of the Boston City Hospital from 1932 to 1939, and physician-in-chief of the Fourth Medical Service from 1936 to 1939. He was named physician-in-chief of the Peter Bent Brigham Hospital in 1938. He was a fellow of the American College of Physicians, the Massachusetts Medical Society and the American Medical Association.

He is survived by his widow and three children.

WAR ACTIVITIES

UNITED STATES NAVY

The following medical officers of the United States Naval Reserve have been assigned to active duty in the First Naval District:

Adams, J. E., Lieut. (JG)
Agnew, J. G., Lieut. Comdr.
Allman, C. H., Lieut. Comdr.
Alt, R. E., Lieut.
Apollonio, H. L., Lieut.
Armitage, H. G., Lieut. Comdr.
Atkins, H. T., Lieut. (JG)
Atkinson, G. D., Lieut. Comdr.

Bakst, H. J., Lieut.
Balch, F. G., Lieut. Comdr.
Barr, J. S., Lieut. Comdr.
Benjamin, L. B., Lieut.
Brown, B. F., Lieut. Comdr.
Buddington, W. T., Lieut.
Burrage, W. S., Lieut. Comdr.

Carmody, J. T. B., Lieut.
Carr, F. B., Lieut. Comdr.
Carson, P. C., Lieut.
Casten, V. G., Lieut. Comdr.
Clancey, D. J., Lieut.
Clapp, W. B., Lieut.
Coffin, W. K., Comdr.
Cordray, D. P., Lieut.
Crandon, J. H., Lieut. (JG)

Daniels, D. H., Lieut.
Davidson, W. B., Lieut. Comdr.
Dawson, W. E., Lieut. (JG)
Dement, D. E., Lieut. Comdr.
Derow, S., Lieut.
Di Cicco, L. M., Lieut. Comdr.
Doyle, J. B., Lieut. (JG)
Drake, E. H., Lieut. Comdr.
Draper, R. T., Lieut. Comdr.
Duncombe, A. L., Lieut.

Eley, R. C., Lieut. Comdr.

Faulkner, J. M., Lieut. Comdr.
Faunce, C. B., Comdr.
Fay, J. H., Lieut. Comdr.
Feldman, S. R., Lieut.
Fleischer, W. E., Lieut. (JG)
Forbes, A., Lieut. Comdr.
Freeman, R. G., Lieut. Comdr.

Gardner, S. N., Lieut. Comdr.
Gibson, S. T., Lieut. (JG)
Glidden, H. S., Lieut.
Goehring, H. D., Lieut.
Golden, S. E., Lieut.
Goodale, R. H., Lieut. Comdr.

Hamlin, H., Lieut.
Hanson, F. C., Lieut. Comdr.
Harris, H. I., Lieut.
Healy, T. R., Comdr.
Hendrix, O. C., Lieut. Comdr.
Houghton, J. D., Lieut.
Hubbard, J. D., Lieut.
Hudson, H. W., Lieut. Comdr.
Hughes, W. N., Lieut. Comdr.
Hunter, F. T., Lieut. Comdr.
Hunter, R. W., Lieut. (JG)
Hynes, E. A., Lieut.

Jacobson, B. M., Lieut.
Johnson, C. I., Lieut. Comdr.
Johnson, C. W., Lieut.
Joplin, R. J., Lieut.

Kennard, H. E., Lieut. Comdr.
Kunian, D., Lieut.

Lamb, G. R., Lieut.
Lamphier, J. A., Lieut.
Lanigan, W. N., Lieut. Comdr.
Leete, E. D., Lieut. Comdr.
Lewis, E. I., Lieut.
Lewis, J. P., Lieut. Comdr.
Lynch, G. W., Lieut.

Macaluso, A., Lieut. Comdr.
Macdonald, D. H., Lieut.
Macdonald, M. E., Lieut. Comdr.
Mahoney, P. J., Lieut. Comdr.
Manning, E. P., Lieut. (JG)
Marshall, J. R., Lieut. Comdr.
Martin, S. F., Lieut.
McCann, D. S., Lieut. (JG)
McCarthy, H. F., Lieut. (JG)
McCarthy, L. J., Lieut. Comdr.
McGinn, S., Lieut.
Merritt, E. L., Lieut. Comdr.
Miller, R. K., Lieut. Comdr.
Mintz, E. R., Lieut. Comdr.
Monks, J. P., Lieut. Comdr.
Murphy, W. F., Lieut.

Nye, L. S., Lieut. (JG)

O'Brien, T. J., Lieut.
Ormsby, E. B., Lieut. Comdr.
Orr, R. B., Lieut. (JG)

Palmer, R. S., Lieut. Comdr.
Peterson, T. H., Lieut. Comdr.
Petrone, F. J., Lieut. Comdr.
Philbrook, F. R., Lieut.
Pollock, B. H., Lieut.
Pratt, T. C., Lieut. Comdr.

Regan, J. J., Lieut. Comdr.
Rooney, J. S., Lieut. Comdr.

Scarcello, N. S., Lieut.
Schwab, R. S., Lieut.
Seeyle, E. B., Lieut. (JG)
Shedden, W. M., Lieut. Comdr.
Shepardson, R. B., Lieut.
Smith, E. C., Lieut. Comdr.
Smith, S. W., Lieut. Comdr.
Solomon, P., Lieut.
Sprague, H. B., Lieut. Comdr.
Stearns. A. W., Comdr.
Stein, C., Lieut.
Stoops, W. A., Comdr.
Stott, A. A., Comdr.
Swan, C. L., Lieut. Comdr.

Thornton, J. P., Lieut. (JG)

Vance, L. A., Lieut. (JG)
Vieira, E., Lieut.
Volk, R., Lieut.

Walker, T. B., Lieut.
Weiner, F. F., Lieut.
White, J. C., Lieut. Comdr.
Wood, H., Lieut. (JG)
Woodruff, L. M., Lieut. (JG)

Yeomans, A., Lieut.

Opportunity for Appointments of Premedical and Medical Students as Ensigns H-V(P), United States Naval Reserve

The Secretary of the Navy recently approved a change in Navy regulations whereby it is now possible for those premedical students who have been accepted for entrance to, and all medical students in, Class A medical colleges to be appointed in the United States Naval Reserve in Class H-V(P), provided they meet the physical and other requirements for such appointment.

Students who are acceptable will be given provisional commissions as ensigns, and it is the policy of the Bureau of Medicine and Surgery not to nominate such officers for active duty until after they have completed their prescribed medical studies and shall have served one year's satisfactory internship in a civilian hospital accredited for intern training, or shall have been accepted as acting assistant surgeons in the Navy for intern training.

On graduation, and when the bureau has been informed of this fact by the dean, commissions as Lieutenant (JG) MC-V(G), United States Naval Reserve, will be issued to provisional ensigns and, after serving their internship in non-naval hospitals, they will be nominated for active duty. Application for, or acceptance of, either a provisional or permanent commission in the Naval Reserve, does not preclude the possibility of applying for a commission in the Medical Corps of the Navy. Persons affiliated with the Naval Reserve are not subject to induction into Army service by action of local Selective Service boards.

Navy regulations require that all applications for appointments in the Naval Reserve be filed with the commandant of the naval district in which the applicant resides. The address of the commandant in each district may be obtained from the dean of the medical college.

Application forms may be obtained from the dean's office or from someone designated by him, on request from the Bureau of Medicine and Surgery, Navy Department, Washington, D. C., or from the commandants of naval districts. When an application form has been properly completed, it, together with the other credentials indicated on the application form, should be mailed to the commandant of the naval district. He will issue instructions for obtaining a physical examination, fingerprints and so forth.

A premedical student must enclose with his application for appointment a statement, signed by the dean of a medical college, to the effect that the applicant has been accepted as a first-year medical student in a Class A school for the next entering class.

It is the understanding of the Bureau of Medicine and Surgery that Selective Service boards will accept a statement from the commandant of any naval district, to the effect that an application is on file, as basis for deferment until the application has received final action.

REPORT OF MEETING

NEW ENGLAND PATHOLOGICAL SOCIETY

The following report of the Executive Committee was presented at a business meeting of the New England Pathological Society on January 13, 1942. The report was accepted, and the recommendations adopted.

* * *

Recommendations of the Executive Committee Concerning the Status of Pathologists during the War Emergency

Maximal war effort requires the maintenance of normal standards of health in the civilian population, and an in-

escapable corollary is the continued efficient conduct of civilian hospitals. The inevitable transfer of medical, nursing and technical personnel to military service and of labor to both industry and military service will make this difficult. A more specific danger is the potential loss of certain highly trained specialists, relatively few in number, who are essential to modern hospital practice. Pathologists and roentgenologists are obvious examples. Even in prewar conditions, the supply was inadequate. Many will certainly be required for both the Army and Navy, and all hospitals will be left either understaffed or completely destitute unless a skeleton group of reasonable dimensions is maintained in each locality and is so organized that its efforts may be rendered most efficient. In an effort to formulate plans for meeting this prospective emergency, the Executive Committee of the New England Pathological Society proposes the following suggestions for consideration.

At the present moment, only general principles can profitably be outlined. Specific recommendations must await more precise definition of the problem and depend on the number of pathologists drawn into the services and on the number, size and location of the hospitals in need of aid. It is to be noted that the situation differs essentially from that which existed in World War I, since at that time, few of the small or community hospitals had pathological laboratories. It is to be hoped that few hospitals under the pretext of economy will revert to the standards of twenty years ago.

Certain premises can be laid down:

(1) Pathologists must be maintained in the larger metropolitan hospitals and in the departments of pathology of the medical schools, to provide for the accelerated medical curriculum. Administrative officers of schools and of hospitals fortunate enough to maintain a pathological staff should be approached, and their co-operation enlisted in solving the needs of the larger communities.

(2) Although much unessential work is demanded of laboratories in ordinary times that can and should be largely eliminated, an irreducible minimum of diagnostic laboratory procedures exists and must be maintained.

(3) Two methods for maintaining this minimum are available:

(*a*) To send the material by mail or messenger to central laboratories where pathologists must be maintained, such as the laboratories of the large metropolitan hospitals, the laboratories of the medical schools and the small laboratories where a pathologist is still on service, including private laboratories.

(*b*) To carry out the technical work locally and arrange a consultative service by which a pathologist will visit at regular intervals to make histologic diagnoses and to supervise and check the technical staff, and by appointment will respond for frozen-section diagnoses and autopsies.

Plan (*b*) service, although obviously preferable, will be much more difficult to maintain. Only a limited number of hospitals can be so provided for, and these must have adequately equipped and staffed laboratories and must lie within a limited geographic area in relation to a central laboratory.

In arranging contracts with hospitals, the following principles should be borne in mind:

(1) It should be assumed that all pathologists called to military service are on leave of absence from their respective posts. Therefore, all contracts of consulting pathologists must be terminable with the return of the original pathologist to civilian life.

(2) The hospitals must pay the cost of services rendered. Under Plan (*a*), this may appropriately be calculated according to the scale of charges authorized by the New England Pathological Society. It is preferable that the fees be paid directly to the pathologist assuming the work and that he, in turn, adjust with his primary employer for the costs of supplies, increased technical and secretarial services and so forth. Under Plan (*b*), the consulting pathologist should receive compensation in the form of a salary proportionate to the work and time involved. The consulting pathologist should not allow the newly assumed duties to cut seriously into the time paid for by his primary employer without negotiating a reasonable adjustment in salary.

(3) The consulting services so offered must fall significantly short of the full service of a pathologist to his hospital. Minimum diagnostic requirements will be met, but the intangible values of constant availability for consultation, for minor administrative tasks and so forth must be sacrificed. It should be possible so to adjust the cost of this limited service that the hospital may, without increased expense, make up to its pathologist in service an approximate difference between his former civilian and his present military salary.

It is proposed that the New England Pathological Society set up a group of regional committees from which any member who expects to enter military service may seek advice concerning his individual problem, and to which hospitals in need of pathological services may apply.

* * *

Subsequent to the meeting, the following regional chairmen were appointed:

Eastern Massachusetts. Dr. Tracy B. Mallory
Massachusetts General Hospital, Boston.

Western Massachusetts. Dr. J. S. Beck
Memorial Hospital, Worcester.

Maine. Dr. Mortimer Warren
Maine General Hospital, Portland.

New Hampshire and Vermont. Dr. Ralph Miller
Dartmouth Medical School, Hanover, New Hampshire.

Rhode Island. Dr. B. Earl Clark
Rhode Island General Hospital, Providence.

Connecticut. Dr. Harry M. Zimmerman
Yale University, School of Medicine, New Haven.

BOOK REVIEWS

Bacillary and Rickettsial Infections, Acute and Chronic: A textbook — black death to white plague. By William H. Holmes, M.D. 8°, cloth, 675 pp. New York: Macmillan Company, 1940. $6.00.

Before the early part of the nineteenth century at least, no man who undertook the discussion of a particular disease failed to include something of its history. Dr. Holmes has resumed that valuable and interesting prac-

tice for each of the maladies with which he deals. Without it, he would still have written an excellent and trustworthy book, for his discussion of etiology, epidemiology, diagnosis, treatment and prevention leaves little to be desired. With it, he has achieved something that for a modern textbook is almost unique. The classic descriptions, of which there are many, are to be cherished. Specifically the maladies considered are plague, tularemia, the rickettsial infections of man, undulant fever, cholera, typhoid and paratyphoid fevers, the bacillary dysenteries, diphtheria, botulism, tetanus, gas gangrene, pertussis, the hemophilic infections, leprosy, tuberculosis, sarcoidosis and the like (considered together in a final chapter). The various sections are closed with adequate bibliographies.

One might add that the book is of convenient size and weight and is delightfully printed. It should be a welcome book for anyone whose practice embraces its matters.

A Research Conference on the Cause and Prevention of Dental Caries. Chicago, Illinois. July 1 and 2, 1938. Sponsored by The Good Teeth Council for Children, Incorporated. 8°, cloth, 178 pp., with 11 figures, 12 tables, 6 charts and 6 graphs. Chicago: Good Teeth Council for Children, Incorporated, 1940. $2.00.

In this small volume, reports of research on dental caries by twelve nationally known investigators are assembled, together with discussions by other research workers. The wit and wisdom of Dr. E. V. McCollum, of Johns Hopkins University, who was the moderator of the conference, illuminate the text.

Of the many factors that may be involved in the etiology of dental caries, five are presented in this volume: the morphology of the teeth, the composition of the saliva, the role of vitamin D, age incidence and the flora of the mouth.

The reports and discussions indicate the complexity of the problem of dental caries but bring out quite clearly the directions being taken by these investigators in their search for new knowledge. The anatomic, chemical and bacteriologic background of dental caries is thoroughly discussed.

For those engaged in any phase of research in dental caries, this book should be a valuable reference volume.

The Endocrine Function of Iodine. By William T. Salter, M.D. 8°, cloth, 351 pp., with 45 tables and 40 illustrations. Cambridge, Massachusetts: Harvard University Press, 1940. $3.50.

This is the first of the current series of Harvard University monographs in medicine and public health. The author, now professor of pharmacology at Yale University, has worked with Harington in England on the chemistry of the thyroid gland; with Means and the clinical group at the Massachusetts General Hospital; and at the time of writing, as both clinician and biochemist at the Thorndike Memorial Laboratory of the Boston City Hospital. He is, therefore, well qualified to present a broad view of the subject.

The book does not give a systematic or exhaustive discussion of the thyroid gland, but takes as its point of departure the nature and functions of body iodine, surveying the thyroid and other endocrines only from the standpoint of iodine. This gives in some ways a rather disconnected story; on the other hand, it brings together for the first time all the available studies of iodine in body economy and endocrine balance, and leaves to the textbooks the conventional approach to the thyroid gland and its disease.

The early chapters review the iodine content of all the body tissues and organs, and of the blood and body fluids. The new technics for determining plasma iodine and its fractions have been of great value. There is a discussion of important iodine-containing compounds, such as thyroglobulin, di-iodotyrosine and thyroxine. Then follows a consideration of the role of iodine in thyroid activity, in relation to the pituitary gland and its thyrotropic hormone, and in the interactions of the pituitary and thyroid glands and the gonads. The newer work on iodine balance is presented at some length, and there is a most interesting discussion of radioactive iodine and its recent uses in the study of iodine metabolism by following "tagged atoms" of radioactive isotopes.

Of interest to clinicians is a chapter on clinical problems, in which ten selected cases are cited to show the application of such new diagnostic aids as total-plasma iodine, the "P" and "T" plasma-iodine fractions and the fecal iodine excretion. One is impressed with the need for meticulous control of all conditions in making these tests, and for caution in their interpretation.

There is an appendix for laboratory workers, in which significant new technics are set forth in detail. An exhaustive bibliography adds greatly to the value of the work.

The Pharmacological Basis of Therapeutics: A textbook of pharmacology, toxicology and therapeutics for physicians and medical students. By Louis Goodman, M.D., and Alfred Gilman, Ph.D. 4°, cloth, 1383 pp., with 126 illustrations and 67 tables. New York: The Macmillan Company, 1941. $12.50.

This large volume covers the pharmacology, toxicology and therapeutics of practically all the drugs now in common use. It is an extraordinary compilation — clear, detailed, accurate, well documented, adequately illustrated and superbly indexed. No book in recent years has so appealed to the reviewer as a fundamental contribution, finely executed. Although the price is not low, the book is well worth its cost. It should be in every medical library and research center, and would be a valuable addition to many physicians in their private libraries. The book will constantly be referred to by any doctor who attempts to keep up with modern trends, and its value to the scientific investigator is great.

The volume is excellently published; the paper is light, but adequate, the illustrations are clear, and the bibliography is easy to read.

A Textbook of Dietetics. By L. S. P. Davidson, B.A. (Cantab.), M.D. (Edin.), F.R.C.P. (Edin. and Lond.), F.R.S.E. and Ian A. Anderson, M.B., Ch.B. (Aberd.). With diet sheets constructed by Miss Mary E. Thomson, S.R.N., and a foreword by Sir John Boyd Orr, M.D., D.Sc., LL.D., F.R.S. 8°, cloth, 324 pp., with 27 tables. New York: Paul B. Hoeber, Incorporated, 1941. $4.25.

The problem of nourishment in some form or other has always occupied the attention of man. This has now become a mass problem because of widespread economic and social dislocations. The introduction of more commercial methods of food preparation is also a source of abnormal nutrition. It is incumbent on every physician to be conversant with a knowledge of dietetics, which is applicable to the recognition of late and frank cases of vitamin deficiencies, problems of malnutrition and obesity and the care of specific metabolic diseases. This book can be regarded as an adequate introduction to the subject. Unfortunately, since it is a British publication, many preparations are mentioned that are unfamiliar to Americans, and the difference in dietary habits is noteworthy. Both considerations lessen the value of the book for Americans.